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COMPLICATIONS OF SPINAL ANESTHESIA*

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IN reviewing the literature on the complications of spinal anesthesia it was found that in most instances phenomena that are part and parcel of the *modus operandi* of the anesthesia, that are but temporary in nature and that despite their unpleasantness are in no way a menace to future health or life are included as definite complications. Inasmuch as these so-called complications, to wit, the fall in blood pressure that is so frequently unnecessarily alarming to the anesthetist or surgeon, the nausea, retching or even vomiting of the patient within a few minutes after the injection of the anesthetic, the pallor, at times associated with a cold, disconcerting clamminess of the skin, and the enfeebled pulse, often barely perceptible, inasmuch as these so-called complications are by far the most frequently mentioned, and since they form the basis of a great deal of the objection that has been raised against the routine employment of spinal anesthesia, it seemed appropriate to present a paper with the subject of complications as its main theme.

A rather comprehensive survey of the literature was undertaken. Over two years of research were devoted to the subject and articles dealing with intraspinal anesthesia since its very inception were studied. The English, French, German, Spanish and Italian journals were searched for material. Inasmuch as the bulk of medical literature finds expression in one of the foregoing languages a rather conclusive symposium was available. In view of the low incidence of complications following the use of intraspinal anesthesia as reported in these journals it seems fair to state that the injection of an anesthetic substance into the subarachnoid space preliminary to surgical intervention has been given the test of time and quantity and has been found to be exceptionally low in either frequent or serious sequelae.

Articles dealing with the subject have been reported on the completion of a series of cases ranging from 100 to 20,000 intraspinal injections. Most frequent mention is made of headache. Less frequently, reference is made to symptoms that are classified as meningeal in origin, being the result of either a meningismus or, even more rarely, a true meningitis. Occasional reference is made to the appearance of neurological disorders as, for instance, paresthesias, myelitis, anal and vesical incontinence, paralysis of the external rectus muscle of the eye, and trophoneurotic disorders ranging from decubitus ulcers to a well-marked paraplegia. Mental aberrations varying from a mild psychosis to pronounced insanity have been recorded as sequelae. Syncope, coma and respiratory failure have been noted. The increasing popularity of subarachnoid injection as a means of inducing surgical anesthesia has stimulated the reports of accidents

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that have occurred as a result of lumbar puncture in itself. Indeed, an appreciable percentage of the fatalities that have occurred immediately after the injection of the anesthetic has been attributed to what some authors have seen fit to designate as "lumbar puncture shock."

HEADACHE

The most troublesome complication of spinal anesthesia is the occurrence of headache. The majority of patients thus afflicted begin to complain as a rule within the first twenty-four hours after the operation. Usually, the headache is either occipital or parietal in location and is frequently associated with some degree of stiffness of the neck muscles. The intensity varies. In most instances the discomfort is so mild that the administration of either aspirin or pyramidon or the application of an ice cap suffices. Rarely is the suffering of such great intensity that the usual analgesics are of no avail. In both the mild and the severe grades the headache is worse when the head and shoulders are raised. Relief is usually obtained in the former type by maintaining the horizontal position.

Headache is not only the most troublesome complication of spinal anesthesia but it is also the most frequent. According to the reports that have appeared in the literature the average incidence ranges in the neighborhood of 20 per cent. As high as 40 per cent, however, has been encountered. Mahler[1] in an experience of 1000 intraspinal anesthesias mentions but 10 per cent of headaches and calls attention to as high a proportion after simple diagnostic lumbar puncture. In the main these headaches disappear within twenty-four to seventy-two hours. The severe, agonizing, persistent type that resists the ordinary analgesics is rare. Indeed, in our own series of over 6000 cases their incidence has been no greater than 600, in other words, 10 per cent.

Featherstone[2] sent inquiries to 100 consecutive patients to whom he had administered spinal anesthesia with the aid of tropococaine. Not one complained of serious disability that in any manner could be charged against the method of anesthesia. Headache and dizziness were the most frequent sources of complaint. Of 70 replies, some degree of late headache was said to be present in 28 cases, that is to say 40 per cent. One man complained; the remainder were women. He adds:

> Astonished by the high incidence of the trouble, I wrote to 20 patients who, under general anesthesia, had undergone operations of a similar nature to those suffering from late spinal headache. There were 16 replies, and in 11 of these the patients had a headache. Thus, of those who under general anesthesia had undergone operation for conditions likely to cause headache, 68 per cent did in fact develop late headache. I think I am justified in blaming the patient's complaint rather than the spinal anesthetic for this late headache. Giddiness did not occur in those patients who were otherwise robust.

In our clinic the administration of spinal anesthesia is not limited solely to so-called poor surgical risks. In fact, save for such short procedures as the incision and drainage of an abscess and the like, subarachnoid block is induced routinely. It has been interesting to observe with what irregularity so-called spinal headache occurs. Frequently, a series of 100 cases, operated upon for varied surgical conditions, comprising all age groups and both elective and urgent conditions, poor risks and doubtful risks, will include but 2 per cent of mild headaches lasting no more than two or three days and amenable to the usual analgesics. In such a series the absence of agonizing headache will be noticeably in evidence. Then, again, a second series may contain as high an incidence as 20 per cent of the mild headaches and may show perhaps 1 or 2 or even 5 per cent of the more severe grade necessitating methods of treatment other than those ordinarily required. We have encountered marked headache that has persisted for more than one week

and that has recurred after temporary relief so that the duration of the affliction may be said to have existed for several weeks in but 5 instances out of a total number of inductions that at the present writing is as high as 6000 cases.

As is to be expected much theorizing has been done in an effort to explain the cause of this type of headache. Among the several ideas advanced are the following:

1. Loss of cerebrospinal fluid through the puncture wound in the dura by seepage into the extradural soft tissues.

2. An increase in the volume of cerebrospinal fluid contained within the subarachnoid space.

The mechanism of occurrence in the first instance has been ascribed to the lack of elastic and contractile tissue in the fibrous dural membrane. It is believed that the puncture hole caused in the dura by the needle point does not close until a fibrin clot forms or normal healing takes place. Castro Silva[3] states that it has been his lot to perform laminectomies eight or fifteen days after a lumbar puncture and to find the tracts of the puncture still gaping, closed simply by non-adherent, epidural fat.

Upon the withdrawal of the needle, the dura and the underlying pia-arachnoid are left in one of two possible relations. Either the openings in the two membranes are superimposed, if the pia is drawn back through the dural opening by the needle, allowing leakage to take place or else the movement of the soft membranes upon each other causes the openings in the respective membranes to be situated at different levels. The force of the cerebrospinal fluid is then thought to compress the pia arachnoid against the dura for an interval long enough to permit the pial opening to close by virtue of its inherent elasticity. If the latter mechanism takes place very little cerebrospinal fluid will escape.

As a result of the considerable seepage of spinal fluid during the first few hours after the operation, there is a disturbance of the intracranial portion of the cerebrospinal fluid. It has been claimed that the latter factor, by removing the watery cushion, allows the brain to sag against the bony framework of the skull. This in turn, supposedly, irritates the dural fibers of the trifacial and the two occipital nerves. Pressure upon the basilar venous plexus diminishes the outflow of venous blood from the cranial cavity and brings about an increase of venous tension. Lowering the patient's head is thought to remove the brain from contact with aforementioned nerves and venous plexus and thus tend to lessen the severity of the headache. Relief is similarly obtained when the opening in the dura has closed, seepage of fluid has ceased, and the normal watery cushion of the brain has been restored.

This type which may be referred to as the seepage type of headache is ascribed ordinarily to the lumbar puncture itself. The second variety of headache, however, is thought to be the result of an increase in the quantity of cerebrospinal fluid. Usually, other symptoms and signs are associated as, for instance, stiffness of the neck, photophobia, and more rarely, paralysis of the external rectus muscle of the eye with or without a concomitant diplopia. This syndrome is described as the postanesthetic meningitis or meningismus. Whereas in the former variety, the spinal fluid pressure may be very low, there is usually found an increase in the pressure associated with an increase in the cells and globulin constituents of the cerebrospinal fluid in those patients with meningismus. Thus, either a state of meningeal irritation is present or else, rather infrequently, a true infectious meningitis. Such headaches do not respond to the lowering of the patient's head.

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demns the larger caliber needles because more fluid can escape through the larger opening and healing is proportionately delayed. We ran a series of over 300 cases with a fine caliber needle and found no appreciable change in the incidence of headache. According to Evans[5] this seepage type of headache is associated with a diminished pressure and he counsels against a further lumbar puncture "as it will make this type of headache worse." Valdone[6] on the other hand, reporting an incidence of 30 per cent (tutocaine anesthesia), after stating that he considered the loss of cerebrospinal fluid as the cause of headache, employed secondary lumbar puncture in 8 cases that failed to respond to caffeine, aspirin or pyramidon and concludes that his contention seemed "to be proved by the fact that the pressure of the cerebrospinal fluid was lowered." An interesting statement is that of Pardee who, after hearing Neustaedter, Hola and Tolstoochow[7] report that they could find no definite relationship between the calcium content of spinal fluid and postlumbar headache, stated that he saw no reason for ascribing headache to leakage since, in his experience, it has been possible to "take out as much as 80 c.c. from the spinal canal and still have no spinal puncture headache even though the patient may get up immediately afterward." After reading such a statement, one becomes rather perplexed upon studying the recommendations of Wells[8] for the technic of inducing spinal anesthesia with what he refers to as anhydrous cocaine.

We quote verbatim from his report as follows:

> From 15 to 25 c.c. more of spinal fluid are then withdrawn from the spinal canal into a glass syringe using gentle traction on the plunger. The amount withdrawn is determined by the patient's reaction, which consists of a progressive headache, generally occipital. In the early part of the series we allowed the headache to go on until the patient uttered an involuntary high-pitched cry, but this has been found to be unnecessary. We now determine the part where headache starts and then continue to withdraw fluid slowly, stopping when we judge that the headache has reached the maximum.
>
> The entire quantity of cerebrospinal fluid contained in the syringe with the dissolved anhydrous cocaine is then quickly reintroduced into the subarachnoid space and "immediate and complete cessation of headache takes place."

It is interesting to note that headache was not frequently encountered by Delmas,[9] one of the foremost French exponents of spinal anesthesia, despite his technic which makes a particular point of first abstracting from 20 to 40 c.c. of fluid, employing barbitage to dissolve the anesthetic substance and increase the diffusion in order to induce whole body anesthesia.

The question of the treatment of headache is discussed from both the prophylatic and curative standpoints.

PROPHYLAXIS

The following recommendations have been made:

1. Use a small caliber needle.

2. Do not allow the patient to move or elevate the head while being operated upon and for several hours after the injection of the anesthetic.

3. Administer sufficient fluids preoperatively. According to Martin-Arbuthnot[10] the patients on the urological ward of the Los Angeles General hospital have their black coffee and all the water they wish to drink before going to the operating room. Those on the general surgical service have their routine surgical preparation and no fluids by mouth on the morning of the operation and it is among the latter group that dull, frontal headaches are encountered. They recommend, therefore, that there be no restriction in the amount of fluids before operation.

4. Avoid the reintroduction of the spinal puncture needle. Careful attention to the technic of lumbar puncture will minimize

dural trauma and thus lessen both the incidence of leakage and the occurrence of irritative phenomena.

5. Do not inject air into the subarachnoid space or puncture the venous plexuses that surround the anterior and lateral aspects of the spinal column. Featherstone[2] remarking that the French have blamed the injection of air, points out that headache occurs after simple lumbar puncture where it is difficult to imagine how air could enter the subarachnoid space. Nor can we ascribe much importance to the few bubbles of air which we frequently inject since from 60 to 80 c.c. and even more are routinely injected in ventriculography without either routine severe headaches or disastrous results. Bagley[11] has endeavored to blame the incidence of headache in intracranial injuries upon the presence of blood in the subarachnoid space. Believing that this might be a clue to the causation of headache, we have paid particular attention to those patients in whom a bloody tap was obtained. At no time did we hesitate to inject the dissolved anesthetic provided that spinal fluid was obtained after the venous plexus had first been punctured accidentally. Such patients did not show a greater incidence of headache.

CURATIVE

1. Administer the usual analgesics first: aspirin, caffeine, phenacetine, pyramidon. Codeine or even morphia may be indicated.

2. Advise the patient to maintain a horizontal position and apply an ice cap to the head.

3. Darken the room, especially if photophobia or diplopia be present.

4. Eliminate all forms of stimulation (including too many visitors that may be productive of unnecessary excitement).

5. Administer 1 c.c. of either surgical pituitrin or from 3/8 to 3/4 of a grain of ephedrine hydrochloride intramuscularly.

6. In the seepage type of headache administer about 100 c.c. of 0.5 per cent sodium chloride solution intravenously. Relief will come within twenty to forty minutes (Evans). Also, force fluids, using a Murphy drip if necessary.

7. Frequent lumbar punctures are advised for the type of headache associated with an increased spinal fluid pressure and meningismus. This procedure is to be resorted to only after relief has not been obtained from diuretics, cathartics and hypertonic saline solution both orally and intravenously. According to Evans[4] the intravenous administration of 10 per cent glucose solution in physiological salt solution will relieve this type of headache if nothing else will. As much as 1000 c.c. may be given and it may be repeated after six to eight hours.

Early in our experience, we adopted one of the procedures that had proved of value in the treatment of increased intracranial pressure. Retentive enemata of 6 oz. of 50 per cent magnesium sulphate soluton were prescribed. Usually, 1, 2 or 3 rectal injections at intervals of four hours were sufficient to relieve the mild forms of headache that did not respond to the ordinary analgesics and that were not severe enough to warrant a secondary lumbar puncture.

Recently, since some of the patients found it difficult to retain the solution for a time sufficient to allow of any therapeutic value and in some instances a mild proctitis developed, we adopted the intramuscular injection of 2 c.c. of 50 per cent magnesium sulphate as supplied in ampule form for the Gwathmey technic in obstetrics. A fair measure of successful results ensued. We accidentally discovered the increased efficacy of the drug when administered intravenously. A patient had already received one retentive enema which she had not retained satisfactorily and which had failed to relieve her of intense headache, stiffness of the neck and marked photophobia. Within several seconds after an intravenous injection of magnesium sulphate, she experienced a sensation of extreme warmth, perspired freely and became quite dry

in the mouth. Two minutes later, she gratefully stated that all her symptoms had miraculously disappeared. Other similar experiences have since established this as a rather reliable agent in our therapeutic armamentarium. Now, when we receive a complaint of slight headache, the patient receives a coal-tar preparation, usually aspirin. Moderate headache is treated by magnesium sulphate intravenously. Severe headache unrelieved by magnesium sulphate is invariably cured by one or two spinal punctures. In one instance, peculiarly enough, a nurse upon whom a cholecystectomy and appendectomy had been performed, cure was not effected even by repeated spinal taps. The empirical use of ½ grain each of anterior pituitary and thyroid gland extracts produced an almost immediate cure.

It seems quite evident that there is still considerable information to be secured regarding the mechanism of the production of headache. Apparently, in most instances the caliber of the needles, blood in the spinal fluid, the injection of air and seepage are explanations which do not hold water. It is evident that in a fair proportion of cases, increased intracranial pressure (however produced) is an important factor.

BACKACHE

Backache occurs in a small percentage of cases. It may be due either to trauma of the soft parts that are traversed by the needle, especially when several attempts to enter the subarachnoid space have been made, or else to a serous meningitis resulting from the wound in the dura and pia, the liberation of a small quantity of blood by accidental penetration of one of the venous plexuses, or the introduction of rust particles from the needle, or even from the presence of several bubbles of air. No matter what the cause may be, the backache is but short in duration and is frequently relieved by placing a pillow under the small of the back.

In our experience, the incidence is approximately 2 per cent. It has never proved very troublesome nor has it ever persisted longer than a week. This period coincides with the time required for the repair of the local tissue-injury. In no instance that we know of has local suppuration resulted, the tissues always being able to complete the reparative process without necrosis. The backache resulting from the induction of anesthesia must not be confused with that following the dorsal recumbent position on an operating table not fitted to conform to the curve of the spinal column. The former quite accurately can be localized to the site of the puncture; whereas the latter is a more diffuse ache.

The only treatment necessary is a local wet dressing and control by coal-tar depressants, although the former has been resorted to in only one instance in our series.

ANAL AND VESICAL DISTURBANCES

Often reference is made to anal or vesical incontinence as a possible complication. Most authors, however, remark that they did not encounter such a state of affairs or else an occasional case appeared wherein reestablishment of the normal functions required but several days to a week. Orth[12] for instance, reports a case of fecal incontinence that cleared up spontaneously after five days and Izquierdo[13] one that persisted for a week. In Bazy's patient[18] the fecal incontinence was due to the fact that sensibility of the anus had been lost. The need of evacuation was experienced as a peculiar sensation in the region of the pubis. Even after the patient had moved his bowels, he still had a sensation of fullness in the rectum.

On the third day after a double inguinal herniotomy under 0.12 gm. of stovaine, a patient of Sourasky[14] developed urinary incontinence, that is, retention with overflow. This was soon followed by occasional fecal incontinence. Fully a year elapsed before complete restoration of functions

appeared. In a second case, an appendicular abscess in a boy of nineteen, incontinence disappeared at the end of three weeks.

Salleras[15] operated upon a boy of seventeen for acute appendicitis who shortly afterward developed a marked vesical retention. The bladder would distend so that it could retain 800 c.c. of urine without any discomfort to the patient. Incontinence resulted from the overflow. Fecal incontinence was also present as well as exaggerated knee jerks, hyperactive tendo Achilles reflexes, ankle clonus and a positive Babinski reaction. This state, however, lasted only three weeks. Salleras advanced the theory that the medullary centers of defecation and urination had suffered damage as a result of a local irritation of the meninges. The latter gave rise to a meningitis which caused a compression of the area either by the increased quantity of cerebrospinal fluid formed in response to the irritation or by the presence of hemorrhage into the subarachnoid space brought about by the trauma of the needle or else damage to the centers resulted from the onset of a sclerosis.

A most interesting case has been presented by Vialard and Darleguy.[16]

A few days after an appendectomy under syncaine intraspinal anesthesia, from 19 to 23 liters of clear urine were passed daily. Kidney function tests were normal. Physical examination was negative except for a sickly habitus. There was a negative urine save for a specific gravity of 1.001 to 1.002. The spinal fluid and Wassermann examinations proved normal. Several levels of hyperesthesia existed to wit, the trigeminal, C 7 to D 1, D 4, D 5, D 8, D 12, and L 1. Deep sensibility was normal.

Signs of adiposis dystrophia genitalia were not evident. The patient acknowledged, however, that he had suffered for some time from a slight trembling which was especially marked in the upper extremities. Following the onset of the polyuria, the patient lost considerable weight. When not eating or drinking because of anorexia, the trembling was exaggerated. It consisted of small, irregular, myoclonic movements. Fibrillary twitchings were absent. The hands were cyanotic.

Roentgen examination of the sella turcica revealed apparently normal findings. And, also, a psychological examination was negative. The initial thirst became imperative and, when unable to satisfy this thirst, despite the fact that nothing was discernible, he complained of a sensation of swelling of the tongue and lips with itching and even painful smarting.

Urinary elimination did not accurately correspond with the intake, although retention of fluid in the tissues did not take place. The rate of elimination was influenced to some degree by position. Thirst was greater at night and the nightly output was from two-thirds to three-fourths greater than the daily excretion. On days when he was unable to leave his bed, however, the daily output was equal to the nocturnal. He complained of intermittent frontal and occipital headaches that were less intense while he was lying in bed. General feebleness and increasing anorexia appeared. Finally, he presented a typical attack of Jacksonian epilepsy with aura in the calf of the right leg.

Upon discharge from the hospital, his urine output was 20 to 23 liters a day and his weight was one-half that on admission.

On examining the family history, it was discovered that there had been a brother who had died as a result of meningitis at the age of three months. The patient himself had suffered from convulsions at the age of three months and some few weeks previous to admission had had a so-called nervous breakdown. Presumably, according to these authors, both had had during infancy a meningitis that caused the death of one and left latent sequelae in the other. The latter might well have been responsible for the nervous manifestations and for the seizure of Jacksonian epilepsy. Furthermore, there quite likely existed a meningeal plaque at the base of the brain.

According to the same authors, the work of Lhermite, de J. Camus, and Roussy as well as that of de J. Camus, Gournay and Legrand has demonstrated clearly that there exist groups of fibers in the tuber cinereum, the one regulating the metabolism of carbohydrates, the other the retention of water in the organism. The nucleus proprius of the tuber cinereum (situated superficially) would be the center of nerv-

ous polyuria. It is easy to conceive of the causation of the infundibulo-tuberien syndrome (nervous polyuria) resulting from a meningeal lesion limited to the base of the brain near the posterior aspect of the tuber cinereum. The lumbar puncture may have reactivated the latent meningitis either through the direct action of the injected anesthetic or more likely by simply disturbing the equilibrium of the dynamics of the cerebrospinal fluid. The intraspinal injection of sterile water or even of normal saline solution is followed by after disturbances as shown by the experiments of Guinard and Koslowski, later confirmed by Stolz and Schwartz.[17]

Bazy[18] reported a case of herniotomy in a man of fifty-nine who for eighteen months after the operation experienced no desire to urinate. When the desire had finally returned, spontaneous contraction of the bladder failed to appear. Catheterization was necessary for the entire eighteen months. After progressive education of the abdominal muscles, catheterization was at last suspended at the end of two years. Still his difficulties were not at an end since, despite his ability to urinate normally at the beginning of his apparent restoration, he was later obliged to assume a crouching posture. Involuntary urination would take place if he neglected to void for from six to eight hours.

The necessity of catheterizing patients postoperatively occurs in 10 per cent of our cases. Of this number 90 per cent need only one or two catheterizations and the remainder void spontaneously after the third day.

In no instance did retention last longer than the stay in the hospital necessary for wound healing. Never did it last longer than ten days, expecting in the instance of a case at present still in the hospital. The patient, a male, forty-seven years old, was operated three weeks ago for carcinoma of the rectum. A bilateral inguinal gland excision and a transsacral extirpation of the rectum were performed. The patient has not as yet voided spontaneously. The urine is clear. The bladder becomes distended so that the patient becomes uncomfortable and requests relief. At the operation there was no evidence of injury to the urethra or bladder wall. This urinary retention is the only complication presented by the patient.

We have given some thought to the possibility of a postoperative traumatic prostatic enlargement as the possible etiological factor and expect to receive more information by cystoscopic examination.

It is our impression that postoperative urinary retention is much more frequently encountered after inhalation narcosis.

Of all the cases presenting this complication, 75 per cent occurred after operations involving the perineum or pelvis or both. We rarely encounter vesical disturbances in head, neck or thorax operations.

It would seem therefore, that invasion of the pelvis either directly or indirectly through the medium of the upper portion of the peritoneal cavity to a lesser degree, predisposes to retention. Of course, perineal operative trauma is readily recoginzed as a responsible factor.

The hypothesis that the anesthesia induction produces a localized serous meningitis and thus disturbs the anal and vesical spinal centers in the lumbar region of the cord is contradicted by the statistical evidence demonstrating the greater frequency of retention following inhalation anesthesia.

In discussing vesical complications it might also be mentioned at this time that, frequently, following the anesthesia induction in a patient just catheterized, upon opening the abdomen the bladder may be found full. It was formerly assumed that the catheterization had been incomplete; but recent observations on kidney secretion following splanchnic section have presented a satisfactory explanation. It is found that splanchnic section or block results in the simultaneous activity of all the kidney glomeruli. Normally, part of the glomeruli rest while others function. With all of them active during any given time, urinary secretion is tremendously increased. Apparently,

glomerular activity is continuously controlled by impulses coming through the splanchnics. Spinal anesthesia which is high enough to block all the splanchnic influences (i.e., roots of the fifth to the twelfth dorsal and first and second lumbar nerves) abolishes this control and increased secretion results.

Treatment of this complication is limited. Catheterization is essential. Stimulation of the lumbar cord by the administration of strychnine and caffeine seems of little value. The influence of pituitrin on the bladder musculature is unreliable. The instillation of glycerine into the bladder has never been of value in our series. We have had no experience with electrical stimulation of the lumbar cord. We have found enemata second only to catheterization as a means of emptying the bladder. Nor do we hesitate to order them the first day after operation excepting in cases of colonic or intestinal resections or anastimoses.

We have had one experience with vesical incontinence directly referable to the anesthesia. This occurred in a female patient operated on for uterine fibroids. For the first eight days, she voided spontaneously. On the ninth day, it was reported that she was incontinent. Examination revealed a distended bladder reaching almost to the umbilicus. Fifty-two ounces of urine were obtained on catheterization. Following this, spontaneous urination reappeared.

No case of fecal incontinence has come to our notice.

OCULAR COMPLICATIONS

The literature contains but few references to the occurrence of ocular complications after intraspinal anesthesia. These are practically limited to paralysis of the extraocular muscles, usually to the external rectus which is supplied by the abducens or sixth nerve. Although ocular disturbances have set in from several days after the injection to as late as the second week after operation, cases have been reported wherein they occurred immediately following the lumbar puncture. As a rule, the complication, although unpleasant, proves to be transitory. After a maximum effect lasting from one week to ten days, the condition shows signs of beginning cure. Occasionally, complete cure does not take place until two months have elapsed.

Abducens paralysis is usually ushered in by a preliminary photophobia. Diplopia supervenes and is homonymous. Outward motion is limited and there is convergent squint. All these symptoms are exaggerated as the patient attempts to abduct the affected eye. Reber[19] reported 5 cases of transient abducens palsy from the service of Babcock. These appeared after the use of ampules of stovaine and tropococaine that had been imported from Europe. The paralysis became evident seven days after operation. Both Reber and Babcock believed that infection followed the employment of contaminated solutions since no similar complication was encountered during the next eighteen years, during which period over 20,000 spinal anesthesias were administered. Valdoni[20] adds 3 cases in 500 inductions with 2 per cent tutocaine. Gagey[21] reports 1 instance in 700; while Warnecke[22] lists 8 in a series of 1800. Gagey's patient suffered from diplopia for two months; whereas the condition subsided completely in Warnecke's patients after the lapse of several days. Two cases were reported by Tits,[23] one four days and the other six days after the injection of 15 cg. of allocaine. Both, however, proved to be transient. Van Duyse fils[24] referred to 4 further cases similar to those of Tits. In one of his patients the paralysis of the abducens appeared shortly after the intraspinal injection but disappeared entirely at the expiration of several days. In another, however, ten days passed before any indication of ocular disturbance became evident nor did the patient begin to show the slightest sign of improvement until two months later. Satanowsky[25] adds one transient case of abducens palsy.

A functional optic neuritis occasionally accompanies abducens palsy. There is a

disturbance of vision, sometimes insignificant, at other times, rather marked. The degree of disturbance is not always proportionate to the degree of changes seen on ophthalmological examination. There may even be complete blindness, though temporary, as reported by Wells.[26] This alarming complication occurred in one instance out of a total of 557 cases by Wells after the use of anhydrous cocaine. Fortunately, the patient recovered completely at the end of five days of grave concern. Functional optic neuritis appeared in one of Tits' patients but vanished after several days.

Reboy[27] states that he has never encountered any case of choked disc as reported occasionally in the literature of spinal anesthesia. According to his views the infrequent paralysis of the trifacial, common oculomotor and external oculomotor nerves, and pressure on the optic nerve (choked disc) can be explained by hypertension of the cerebrospinal fluid consequent to a serous, toxic meningitis caused by the injected anesthetic agent. He recommends that decompressive lumbar punctures be instituted as soon as these signs appear.

Drooping of the left upper eyelid (the result of a paralysis of the branch of the oculomotor nerve which supplies the levator palpebrae superioris) was reported by Wells.[26] This disappeared at the end of two weeks and was encountered in but 1 case. Martin and Arbuthnot[28] in a review of over 6000 cases operated on under spinal anesthesia at the Los Angeles General Hospital mention but 1 ocular complication, an inability to move the eyeballs without causing intense pain. This was accompanied by numbness of the feet which persisted for two days, severe headache and neck pain that lasted for three days.

When paralysis of the external rectus occurs, it is usually unilateral, and as frequent in the right as in the left eye. It does however, occur bilaterally as reported by Lang,[29] Baisch,[30] Goetermann[31] and Landow.[32]

The majority of authors who have reported ocular complications after intraspinal anesthesia point out that one must not be too prone to consider the anesthetic at fault since similar disturbances occur at times following lumbar puncture alone. Guillain and Alajouanine and La Grange[33] have observed papillary hyperemia in patients who displayed other signs and symptoms of lumbar puncture intolerance, such as, headache, nausea and vomiting. Examinations previously showed normal eyegrounds. The papillary picture was remarkably constant and persisted several days after the subsidence of the other symptoms. In patients who complained of headache, vertigo and nausea of only several days' duration the ophthalmological picture showed only a congestion of the papilla without all the signs of a true papillitis. When no evidence of lumbar puncture intolerance was obtained, no ophthalmological changes occurred. Guillain and Alajouanine and La Grange believe that the papillary changes are caused by an intracranial, paroxysmal hypertension. This can be explained by a hypersecretion of the choroid plexus caused by the removal of cerebrospinal fluid or other modifications in the hydraulics of the fluid. This hypertension may impose an obstacle to the return circulation or it may produce symptoms of localized arterial hypertension.

Thus, the ocular complications of spinal anesthesia must be separated from those incident to the lumbar puncture. We have had the average experience, namely, 1 case of diplopia in 500 inductions, and all cleared up within two weeks.

The explanation usually given of the mechanism by which the sixth nerve becomes involved postulates an edema of the brain consequent to the injection. The long course of the sixth nerve at the base of the brain allows for relatively more stretching. This, it is believed interferes with its function. As the edema subsides, there is a recession of symptoms. If this explanation be valid, why does the diplopia frequently occur four, five or six days

after the injection? Why are there frequently no other symptoms of cerebral edema?

Another explanation advanced is that the sixth nerve is particularly susceptible to the effects of the anesthetic. While there is some evidence that different nerves are variously affected by anesthetics, why, again, the delayed activity? And why not a more uniform occurrence of this complication?

In the treatment of this condition, the element of time is the most important factor. Patients may be confidently assured of complete cure. Rest in the recumbent position is helpful.

Another complication is pain in the eyeballs on ocular movement. This is also best controlled by eye rest and depressants.

We have had a few cases of photophobia. These were associated with meningismus, and lasted no more than one week.

MENINGISMUS

The incidence of meningismus encountered by us is 1 in 600. It is usually ushered in on the day following the anesthesia induction with headache. Dizziness, stiffness of the neck, occasional inequality and sluggishness of the pupils, photophobia and eyeball pain follow. The symptoms are aggravated by raising the head and movement of the body.

The cerebrospinal fluid shows the picture of an aseptic meningitis. The symptoms last for two or three days and then subside spontaneously.

These patients are not relieved by coal-tar derivatives nor even by codeine. Magnesium sulphate by enemata or intravenously and spinal puncture are the only effective measures. They are used here as advocated above in the treatment of headache, the underlying principles being identical.

MENINGITIS

The occurrence of a true, purulent meningitis as a complication of intraspinal anesthesia is a rarity. Despite the warning of certain authors that subarachnoid block is contraindicated in sepsis, numerous cases are on record wherein purulent meningitis did not result. Indeed, but 9 cases in all the hundreds of thousands of inductions over a period of over twenty years have been reported in the literature. Of these, only two resulted fatally. However, the data contained in the reports were so meager that it is possible that not all the cases cited had a true purulent meningitis.

During the first twenty-four to forty-eight hours after operation, the symptoms of headache, stiff neck, restlessness, ocular aberrations and increased temperature make their appearance. Shortly afterward the characteristic symptoms and signs of a purulent meningitis are in evidence. The spinal fluid is turbid, the globulin and cell findings are raised, and there is a variable degree of elevation in the pressure of the cerebrospinal fluid which contains organisms.

Cases of true purulent meningitis have been reported by Wertheimer,[34] Senge,[35] and Sanchis-Banus.[36] Boros[37] reported a case which he described as a case of cystic, purulent meningitis following lumbar anesthesia induced with novocaine. It was his only case in a series of 1439 induction over a period of eighteen years, during which he encountered 25 per cent of headaches. His patient was a twenty-five-year old man, operated upon for a right, incarcerated inguinal hernia. Twelve hours after operation, the patient complained of severe headache, became restless and lost consciousness. At the end of three hours all the cardinal symptoms of meningitis were noted. Boros administered 40 per cent urotropine solution intravenously. Upon lumbar puncture a sterile, turbid fluid under high pressure was obtained. He repeated his injection of urotropine and gave in addition 10 c.c. of trypaflavine intravenously. The symptoms gradually regressed and by the sixth day had ceased entirely. The novocaine was examined and found to be sterile. Boros quotes a similar case in the experience of Pautrier. Silva[3] adds 2 fatalities occuring a few minutes

after the intraspinal injection, the spinal fluid being very turbid in one.

Meningitis has occurred after diagnostic lumbar puncture alone. Symonds[38] reported a case that recovered after the institution of irrigation of the subarachnoid space. Sonnenschein,[39] however, presented a case of meningitis following diagnostic lumbar puncture that terminated fatally.

It would seem, therefore, that meningitis following intraspinal anesthesia need not necessarily result from the use of contaminated anesthetic agents, or ensue in a patient suffering from septicemia consequent to the establishment in the meninges of a *locus minoris resistentiae* brought about by the trauma of the lumbar puncture needle.

We have operated upon at least 30 patients in whom there were positive blood cultures without the production of a meningitis. It is our belief that such cases do not offer a contraindication to the use of spinal anesthesia.

Five years ago, seven days after an appendectomy in a sixteen-year old girl, classical symptoms of meningitis developed. Examination of the cerebrospinal fluid revealed the meningococcus as the causative agent. The biological characteristics of this organism ruled out the likelihood of any but a coincidental relationship of the meningitis to the anesthesia. This case terminated fatally after three weeks.

In a recent unpublished report of 496 spinal anesthesias from a local institution there was 1 fatal case of meningitis due to a pyocyaneus infection.

It must be remembered that suppuration at the puncture site is a definite contraindication to spinal anesthesia. Van Lier[40] reported a staphylococcus meningitis as a result of passing a needle into the subarachnoid space after traversing an unsuspected abscess in the sacrospinalis muscle. The cerebrospinal fluid was but slightly contaminated and the patient recovered.

The treatment of this complication needs no discussion here.

INJURIES TO NERVES

Nerve injuries following spinal anesthesia are fortunately rare. Furthermore, they are usually transient in nature. Exceptionally, symptoms and signs may exist for many months and cause not only great discomfort and annoyance to the patient but also considerable chagrin to the surgeon.

Damage to the spinal cord or to the roots may result in anesthesia, paresthesias, trophoneurotic changes leading to atrophy of muscle groups, decubitus ulcers, sharp, lancinating pains along the course of nerves, and transient anal or vesical incontinence. If more severe damage has taken place, transient paresis may be replaced by permanent paralysis, although the latter is even more rare than the sequelae of the milder forms of nerve irritation or trauma. It is difficult to damage the nerve bundles even when they are held free in one's fingers and attempts to penetrate them with a needle under direct vision are made.

Ramentti[41] reports a fatal case in a man, aged sixty-five years, who was dismissed on the twelfth day after radical operation for hernia, and on the fourteenth day after leaving the hospital developed meningismus with paresis, paraplegia and other symptoms of cord disease, with disassociation of sensibility, septicemia, splenomegaly and jaundice. Death appeared seven weeks after the onset of the symptoms. Devraigne, Suzor and Laennec[42] described a most interesting experience. Cesarian section was performed upon a multiparous woman of twenty-six because of the presence of a placenta previa. Two c.c. of a 5 per cent novocaine solution were employed. Heaviness of the lower extremities appeared four days after operation. On the following day there was a complete flaccid paralysis of the lower extremities. The knee jerks and the tendo Achilles reflexes, and sensation were absent. The trunk muscles and upper extremities were equally paralyzed. The olecranon and radial reflexes were also abolished and paralysis extended as high as the muscles

of the neck where it was less marked. There was no paralysis of the eye, tongue or soft palate muscle; nor was there any vesical or anal incontinence. Headache was conspicuously absent. The condition was thought to be due to a more rapidly spreading poliomyelitis than that usually seen in the ordinary infectious variety. On the following day, very little paralysis was present, and on the day immediately after, the quadriplegia was almost all gone save for slight weakness of the muscles of the lower extremities. The patient was discharged as cured eighteen days after operation.

Out of a total of more than 1100 cases, Case[43] reports one nerve complication which occurred on the service of a colleague, J. M. Nielson. A perineal repair was done on a thirty-six-year old woman. As the anesthesia was beginning to wear off, the patient began to complain of severe pain in the tip of the coccyx and sacral region. This continued unabated for two weeks and was relieved temporarily by heat and not in any other way except by opiates. With the pain there appeared a saddle-shaped area of total anesthesia in the perineum and buttocks, and rectal and vesical insensibility, the patient losing control and becoming incontinent.

When seen two weeks after operation, the foregoing was confirmed; but there was in addition hyperesthesia in the first sacral and second sacral areas on the left and in all the lumbar segments on the right. The lumbar segments escaped on the left; while the first and second sacral segments escaped on the right. The pain was greatly increased by raising the trunk, straining coughing or sneezing. A diagnosis of cauda equinae lesion was made, probably hemorrhagic. Improvement had been steady but slow. At this writing, five weeks after the onset, there is marked sensitiveness in the area formerly totally anesthetic. The other segments have returned to function but the patient walks clumsily. The final healing in this case was complete and there is at present no complaint whatsoever, nor any objective sign of nerve disturbance.

Orth[12] reported no nerve complications in a series of 1400 lumbar anesthesias and Warnecke[22] none in 1500 inductions. A case of paraplegia of the lower extremities as described by Lemoine[44] is of interest. No anesthesia resulting after the apparently subarachnoid injection of 12 cg. of allocaine for a varicocele operation, he resorted to a local novocaine infiltration. On the same evening, there appeared a paraplegia of both lower extremities, with the sensation of pins and needles in the feet. Micturition was normal. The planter and abdominal reflexes were intact but the knee jerks were absent. On the seventh day after operation anesthesia of the legs was no longer present and motility was almost normal. Lumbar puncture on the second day after operation had elicited a clear spinal fluid with no signs of meningeal irritation.

Bazy[18] operated upon a fifty-nine-year old man for hernia. Soon after operation, the patient complained of pain in the limbs similar to cramps and weariness. Complete loss of sensation in the entire gluteal and genital regions followed. The gluteal muscles, especially, were hard on palpation; nor was their elasticity hardly restored after three years. Increased knee jerks were likewise present.

In a small series, no more than 100, under novocaine anesthesia, Izquierdo[13] noted 1 instance of paralysis of the lower limbs that lasted for an entire month, 1 case of anal incontinence of a week's duration, and 1 case of marked necrosis. None of these cases were described in detail. Decubitus ulcers have been reported by Bilancioni,[45] Fischer,[46] Klein,[47] and Sudeck.[48] Nonne and Demme[49] encountered a case of degenerative myelitis in a man of fifty-nine, operated upon under tutocaine anesthesia, who later expired. Three instances of paralysis of the external popliteal and sciatic nerves were noted in a series of 2207 rachianesthesias by Cotte.[50] Babcock's experience[51] is particularly interesting. On 11 different occasions, spinal anesthesia was induced in one of his patients without any evidence of nerve injury. In 1 case, however, he produced a marked radiculitis by the

accidental injection of a nerve root. Lee[52] who has seen no cases of injury to the spinal cord states that Weigelt[53] adds two additional cases to two that he had previously reported. Paraparesis occurred in a woman fourteen weeks after spinal anesthesia. This was eventually followed by complete recovery. In the other patient the paralysis followed the intraspinal anesthesia twenty-seven months after its injection, with a fatal ending. Autopsy showed obliteration of the subarachnoid space in the thoracic and lumbar regions. The obliteration was caused by chronic inflammation and fibroid thickening of the meninges. Secondary changes in the spinal cord were due to deficient blood circulation, and to compression. It seems to us that one should be careful before ascribing such a result occurring so long after to the subarachnoid injection.

Organic changes rarely come on in after-life following a previous spinal anesthesia. Occasionally, patients have resorted to the law in an effort to collect compensation on such a basis. According to Evans[5] only 2 cases appear in the literature where, after careful neurological examination, such a claim, following lumbar puncture, might be justifiable.

Direct injury to the cord may be occasioned by high injections. In the 496 cases cited under meningitis reported from a local hospital there was one fatal paraplegia following an intraspinal anesthetic administered between the eleventh and twelfth dorsal vertebrae. It is, therefore, of interest to recall that Jonnesco[54] the foremost exponent of high injections, puncture being practiced at a level as high as the cervical region, has experienced no serious sequelae and no complications in 6200 rachianesthesias.

Three years ago, we did a tracheloplasty, perineorrhaphy, Simpson-Gilliam ventral suspension of the uterus and appendectomy on a twenty-six year old patient. Eight days postoperatively, she developed a weakness in her left quadriceps femoris group which became progressively worse so that, two weeks after operation, she could not raise her leg off the bed. She could not walk for three months. Finally, power was slowly restored to the muscles and at the end of a year, her thigh was as strong as before the operation. In our opinion this complication was due to a nerve root injury occasioned during the spinal tap, despite contrary opinion advanced by consultants, who believed the condition to be due to unexplained trophic disturbance.

We have had 2 other cases of weakness of the quadriceps group lasting only four to five days.

There have been occasional cases of tingling of the lower extremities, anesthesia of the buttocks, itching about the anus, all lasting less than three days. None of these complications presented any serious aspects.

PSYCHIC DISTURBANCES

Much has been written concerning the psychological aspect of spinal anesthesia. Indeed, some authors object to any form of anesthesia, not excluding spinal, which allows the patient to retain consciousness during the operative procedure. This does not apply, of course, to some minor condition for which the public has been taught to expect local infiltration.

Lee[55] in discussing Schultz' report of 2251 cases from the Lankenau Clinic refers to his experience in the Department of Mental and Nervous Diseases, of the Pennsylvania Hospital, where the mental chaos of certain types arising during an operation under some form of local anesthesia has been followed by various grades of mental catastrophe.

Izquierdo[13] mentions 1 case of insanity that cleared up completely after a month's duration. A case of postoperative psychosis was reported by Bianchi[56] that occurred in a young man who had been a hard drinker. Following a herniotomy, an acute psychosis with hallucinations set in and lasted for two weeks. Such a sequel to operation, however, has been encountered under other forms of anesthesia among patients long addicted to the use (or, rather, abuse) of alcohol.

Our experience has revealed very few instances of psychic aberrations following spinal anesthesia. One case stands out clearly in our memory, to wit, a young man, thirty-two years of age, operated upon for a floating kidney. He had been markedly apprehensive over his condition for months before submitting finally to surgical intervention. Having discussed spinal anesthesia with several of his friends who had been operated upon by this method, he did not approach the event with any undue fear. While he was in the hospital, complete collapse of his left lung appeared but cleared up entirely before discharge. Shortly afterwards, he began to complain of a sensation of water running down along his right side, the site of the nephropexy, from the upper margin of the wound down along the right lower quadrant of the abdomen and along the anterior and inner aspects of the right thigh. A thorough physical examination which included several complete genitourinary and neurological investigations failed to reveal any organic cause for the paresthesias of which he complained. At the present date, approximately six months after operation, he still feels these sensations at frequent intervals and has become a well developed hypochondriac, indeed, to such an extent, that he is unable to maintain a steady rate of employment. Lately, we occasionally encounter a psychosis after spinal anesthesia; but only in neurotic or apprehensive patients who have received sodium amytal intravenously previous to operation and in whom the psychosis is directly referable to the sodium amytal. At times, delirium, hallucinations and an acute maniacal state result, to disappear completely within a few days. From straight spinal anesthesia, however, with the exception of the case cited here, we have had no psychic disturbances.

Another possible complication is that resulting from the application of heat to the lower extremities after the patient is returned to bed and before the anesthesia disappears. In one of our cases a hot water bag so applied produced a serious burn of the plantar surface of the foot.

Occasional instances of coma (Coulin)[12] and of recurrent syncopal attacks (Izquierdo)[13] have been reported in the literature.

Pulmonary complications following spinal anesthesia are certainly less frequently encountered than after inhalation narcosis. From January 27, 1929 to September 23, 1929 we operated on 1819 patients. Of these 1010 were done under spinal anesthesia. The remainder belonged to the type of minor operation in which it was not deemed worth while to enter the subarachnoid space, to wit, incision of subcutaneous abscess, excision of sebaceous cysts and superficial lipomata, and the like.

In these 1010 cases which incidentally remained in the hospital ten days or more, there developed 3 cases of pneumonia, 11 of bronchitis, 4 of dry pleurisy, and 1 massive collapse of the lung. There were no fatalities. These cases were collected during the summer and winter months and represent a fair average of weather conditions. Compared with the report of Cutler and Hunt[58] in which 55 pulmonary complications with 11 deaths were noted in 1562 operations under inhalation anesthesia, spinal anesthesia seems preferable.

If there be anything in the hypothesis that inhalation anesthesia by its irritating effect on the tracheobronchial mucosa predisposes to pulmonary infection, block anesthesia is certainly indicated in order to lessen lung complications.

In reviewing the postoperative experience of a large number of patients operated on under this form of anesthesia one is struck by the comparatively slight amount of nursing care necessary to keep them comfortable. From this standpoint alone, one receives the decided impression that spinal anesthesia is followed by fewer annoying sequelae. On the whole, the relative values of spinal and inhalation anesthesia can be gauged only by clinical impressions gleaned from a comparison of an extensive series of cases. Our opinion needs no further expression.

[Note: For references see authors' reprints.]

PERITONITIS FOLLOWING ABDOMINAL SURGERY*

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POSTOPERATIVE peritonitis is a contingency that should not be allowed to occur, but like many things that ought not to be, it will occasionally happen. It is only natural that we ask ourselves, Why? Following abdominal surgery peritonitis may result from infection present before the operation or it may be due to the operation itself; in other words, either an external or an internal factor or both factors may be responsible for this undesirable postoperative event.

Let us first consider the external factors. These may be summed up in the words aseptic technic or rather the lack of it. As we all know, the instruments and the hands of the operating staff may be the source of bacteria and the agency of their dissemination. These factors are avoidable by careful technic. It goes without saying that all instruments, gauze, ligatures, etc., should be aseptically prepared and should be kept sterile. In the Lankenau Clinic we have a detective in the person of the bacteriologist who at any time and as often as he wishes takes smears and cultures of the hands of the operating personnel from the surgeon down. In fact there is a pleasant rivalry among us as to the best record for this personal asepsis.

The second external factor is the skin of the abdomen of the patient. Staphylococcus we know is always present in the skin and is not an infrequent finding in postoperative peritonitis. Therefore an essential part of operative asepsis is careful preparation of the patient. These are mechanical factors. They are controllable and should never be the cause of postoperative peritonitis. But not all external factors are mechanical and controllable: for example, penetrating wounds of the abdomen. While proper technic, as a rule, provides against this type of infection, it cannot always be controlled since much depends upon the conditions prevalent at the time of the injury. But naturally every effort must be made to get as aseptic an operative field as circumstances permit.

Now as to the intra-abdominal factors, or infection present at the operation. This comprises the main point of our discussion. As a rule the infection resides in the operative field and its vicinity. It may however reside in a distant focus. The defensive mechanism of the body may be so weakened by disease that organisms from a distant focus may be released by the operative act and find a fertile soil in the peritoneum. The lesson carried by this possibility is preoperative attention to oral hygiene, the respiratory tract, etc., in fact all precautions that make for safe surgery. The local intra-abdominal sources for possible dissemination are numerous and varied. I need but mention two of the more common ones: encysted collections of pus from an inflamed appendix or from a pelvic abscess. The prevention of peritonitis in these cases is both a matter of diagnosis and of operative technic. Preoperative determination of the presence of an abscessed appendix should lead the surgeon to take the first step in preventing contamination by making the proper approach, an extraperitoneal approach when this is possible. In fact in all recognized infected cases the incision should be so planned as to enable the surgeon to deal with the infected focus without invading healthy tissue. This means also the proper placing of gauze pads, using sheets of rubber dam, providing proper drainage, etc. There are, of course, no rules for these procedures. Their use is a part of surgical judgment and their efficiency is established by the high or low incidence of postoperative peritonitis in the records of each individual surgeon.

An encysted collection of pus in the

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majority of cases of appendicitis fortunately is in the lower right abdomen, and can be evacuated by an extraperitoneal approach; where the collection is high up, lateral and posterior to the base of the cecum and colon, the extraperitoneal incision, carried well out and dividing the muscles in the line of the skin is also less likely to be followed by postoperative peritonitis than when the incision is made over the most prominent part of, or mesial to the swelling. After the abscess cavity has been evacuated and no rivulets of pus are seen emptying into the cavity, and the appendix is neither visible nor palpable, nothing more than drainage of the cavity should be done. It is our practice to pack the cavity lightly with loose gauze wrung out of a 1:20 carbolic acid solution. When the appendix can be seen and felt and can be removed in its entirety without danger of breaking through the limiting wall that separates it from the preperitoneal cavity, it is removed and the cavity packed with loose gauze. When the limiting wall is broken through and especially if there is a small amount of pus at the base of the appendix, the cavity is packed at once, and after separating the parietal from the visceral layer of peritoneum around the inner circumference of the cavity, we lift up the abdominal walls and the underlying parietal peritoneum, introduce a large sheet of rubber dam and follow this by the introduction of one or more large abdominal pads and smaller pads, if necessary, to wall off and thoroughly protect the surrounding peritoneum. The appendix is then removed and the cavity packed. This is frequently done in our clinic with good results, which means no postoperative peritonitis, and as a rule recovery. Where the walls of the encysted collection are made up entirely of coils of small intestine, the risk of subsequent peritonitis is greater. If we are able to recognize the condition we hesitate to do an early operation.

In case of a pelvic encysted collection, if it is low down the incision is made, in the male through the rectum, and in the female through the roof of the vagina, posterior to the cervix. If the pelvic collection is located in the median line the incision is made above the pubis after the bladder has been emptied by catheter. In a pelvic abscess resulting from disease of the uterine appendages, the approach is best made through the vagina. But if there is any uncertainty about the state of affairs, that is, if there is danger of puncturing a coil of bowel, we open the abdomen in order to ascertain the topography and then make the approach through the vagina and close the abdominal wound.

A potential cause of postoperative peritonitis is found in operations on the intestinal tract in which the gut, with its rich bacterial content, has to be incised. Here also the careful planning of the operation and meticulous care to avoid contamination by spilling the intestinal contents will prevent the undesirable postoperative complication we are discussing. In operating an intestinal obstruction when one is not absolutely sure that the obstruction has been relieved there should be no hesitancy in turning out the entire small intestine so that it and the large intestine can be thoroughly examined. No harm need accrue from the procedure provided the operative field is sterile and the intestines are well covered with large warm gauze pads.

The foregoing are some of the more common causes of peritonitis after abdominal surgery. They can be recognized before or at the operation and with judgment and technic can, to a large extent, be so handled as to provide for an aseptic recovery. There are, however, a few potentialities which cannot be foreseen. Fortunately, they are rare. Among these may be mentioned an infected blood clot as in a ruptured extra-uterine pregnancy. In these cases it is always our practice to cleanse the peritoneal cavity of blood clots. Failing to do this runs the risk of the clot or clots becoming infected by migration of microorganisms through the walls of

the intestine. This applies likewise to intraperitoneal hemorrhage from any cause. Other cases may be attributable to overlooked intestinal obstruction due to incomplete primary operation for obstruction. For example, snaring of a knuckle of bowel into a rent in the mesentery, or into a congenital hole, or a loop of bowel that has become engaged between the margins of the opening in the transverse mesocolon through which the stomach is drawn in making a posterior gastroenterostomy and where the lesser peritoneal cavity has not been completely closed, when stitching the margins of the opening in the transverse mesocolon to the wall of the stomach immediately above the anastomosis.

Bile leakage after gall tract surgery causing a bile peritonitis also occasionally occurs and sometimes can be traced to unrecognized anomalies of the bile ducts; or to slipping of the ligature in the cystic duct, or to incomplete closure of the gall bladder bed after cholecystectomy. These are favorable cases for re-operation if not allowed to linger too long.

With these briefly stated facts in mind, it is easy to recognize that abdominal surgery has its pitfalls, which in most instances, however, can be avoided. It is a trite saying that the best way to treat postoperative peritonitis is to prevent it.

As already indicated the most important step in the technic of abdominal surgery where infection is present is to avoid contaminating the peritoneum around the infected area. This is the surgeon's greatest concern. What is the best way to guard against spreading infection? First, its recognition by inspection, color and odor; secondly, by taking smears of the area around and distal to the operative field, having them examined and immediately reported upon. This takes but a few minutes, provided the proper equipment is at hand, which includes above all a competent bacteriologist working in a room adjoining the operating theater. This is one of the operating surgeon's greatest assets. I realize that this equipment is not always at hand, but that fact does not alter the desirability of having it. The report of smears of the area distal to the site of infection is a guide as to how far to carry protection, as well as to the amount and kind of drainage to use and where it is to be placed so as best to serve its purpose.

If in the absence of these precautions or in spite of them, peritonitis does set in, the treatment is the same as for the preoperative inflammation, that is, providing for localization by the usual measures of complete anatomic and physiologic rest and re-operating at the most opportune time. While some surgeons advocate immediate re-operation, this is not our practice, except where the operation has been done for a visceral injury. If, as sometimes happens, the patient on the first or second day appears not to be doing well, prompt re-operation is indicated. By not doing well, we mean, of course, presenting the usual signs and symptoms of peritonitis, rigidity, hyperperistalsis, tenderness, etc. With the patient under the careful constant observation of a well-trained interne, the time for operation can be better seized than in the preoperative case when peritonitis has, as a rule, already set in when the patient is brought to the hospital. While the postoperative case has this advantage, it labors under the great disadvantage of the effects of the first operation and it is only by superhuman efforts and good fortune that recovery takes place. Therefore the trite dictum cannot be too often repeated: The best way to treat postoperative peritonitis is to prevent it.

CONCEALED HEMORRHAGE FOLLOWING OPERATION

DIAGNOSIS AND TREATMENT*

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HEMORRHAGE following laparotomy is fortunately an infrequent complication. The precision of technical procedures and more accurate diagnosis have lessened the possibilities of postoperative hemorrhage. It is, however, still a complication of magnitude and one that should be considered after some types of abdominal surgery. There are certain complications which occasionally follow any kind of laparotomy: shock, gastric dilatation, hemorrhage, cardiac failure and embolism. These are directly due to anatomical causes and when they occur within the first twenty-four hours after operation are sudden and tragic. During the second twenty-four hours, concealed hemorrhage may well be a possible complication and still later hemorrhage may occur as a secondary development of abdominal sepsis. True shock is, however, rare. At the present time the infrequency of shock is due largely to the preparation of the patient by means of a dry technic rather than by the prolonged washing and scrubbing of former days when the patient frequently lay in a pool of wash water and suffered varying degrees of bodily refrigeration.

Bleeding into the abdomen after operation, where the abdominal wound has been sutured without drainage, is almost invariably associated with intense abdominal pain. The escape of blood into the peritoneal cavity induces very definite clinical signs of chemical peritonitis. There is more than the accustomed postoperative rigidity, the abdomen is fixed and the patient can accurately localize the area of pain because it is due to the escape of fluid into a localized portion of the abdominal cavity.

After certain types of operation, particularly where gauze drainage has been employed, such as the tamponage of the bed of the board ligament after removal of an intraligamentous cyst or from the parenchymatous oozing that comes with the evisceration of an inflammatory mass, there is the rapid loss of body fluid as the result of serum drainage and the clinical condition resulting therefrom is very apt to simulate hemorrhage. The patient is somewhat pallid, the pulse is rapid and in a cursory way the clinical picture does suggest the general appearance of the patient who is suffering from intra-abdominal hemorrhage. This condition, however, should not be confused with hemorrhage. The patient has a tachycardia from loss of body fluids, but there is no change in the blood color or in the red cell count. Furthermore if the gauze drainage is pinched between the thumb and first finger there will be a moist impression with no cobweb-like fibrillae of fibrin formation. If there is a real loss of blood upon pinching the gauze between the fingers there will remain upon them cobweb-like fibrillae of fibrin and blood.

Hemorrhage as a postoperative complication may be roughly divided into two great groups: (1) cases in which hemorrhage may be occasionally expected or anticipated, and (2) cases in which hemorrhage is not to be expected or anticipated. Surgical intervention in the right upper quadrant in which the pancreas may be infected or injured are operations that contribute par excellence to the liability of hemorrhage. There is an increased tendency for hemorrhage in any disturbance of the pancreas on account of the unusual blood supply but more particularly from the escaped pancreatic juice with its

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marked lytic properties causing erosion of adjacent or contiguous blood vessels. Whenever jaundice is present there is always the possibility of concealed hemorrhage. It is noteworthy that after operations on the common duct there is a distinct tendency for this complication to occur. The effect of jaundice is essentially chemical, not infectious, and hemorrhage is one of its most frequent manifestations. It is interesting to note that in approximately 75 per cent of the cases that die after operations on the common duct there is found at autopsy an intra-abdominal collection of fluid blood varying from 300 to 800 c.c. and in fully half the cases where death takes place postoperative hemorrhage is an important factor in the production of death.

There is also an increased element of intra-abdominal hemorrhage after hysterectomy, after enucleation of tumors from the bed of the broad ligament. The hemorrhage that sometimes ensues after hysterectomy is usually rapid, characterized by very great escape of blood and the clinical signs of hemorrhage are dramatic and rapid. In the hemorrhage that occurs from oozing from the bed on the broad ligament or after enucleation of inflammatory masses, there is apt to be capillary bleeding from many bleeding points which individually are negligible but which collectively represent a great loss of blood. This type of bleeding occurs from raw areas of intestines, or where large portions of the peritoneum have been denuded as in the separation of inflammatory or postoperative adhesions. Let it be remembered that the omentum contains no muscle tissue and every bleeding point on the omentum is apt to continue to bleed unless it becomes adherent or agglutinated to adjacent peritoneal surfaces or ligated.

The diagnosis of concealed hemorrhage rests upon the ascending pulse rate. In all laparotomies where there is the possible chance of hemorrhage being a factor, such as gastroenterostomy, resection, hysterectomy, enucleation of intraligamentous cyst, anastomotic operations on the bowels, operation in the presence of acute inflammatory tissue, a very excellent precaution is to take half-hourly pulse determinations and have them charted; an acceleration of the pulse rate from half hour to half hour, over a period of time, is especially significant. A successive fall in the red blood count and in the hemoglobin are of value when hemorrhage is suspected but it will seldom be carried out as a routine postoperative measure.

After almost all clean cases of abdominal surgery there is a rise of temperature for the first twenty-four to thirty-six hours. This is a natural reaction, representing the so-called surgical, traumatic or operative fever. There is a definite parallellism in the relationship between temperature, pulse and respiration. Theoretically, for each degree of fever one can expect an increase in the pulse rate by ten, an increase in the respiratory rate by two, and this parallellism is ordinarily preserved. If hemorrhage in any great degree has taken place there is a loss of this definite parallellism and the pulse rate is increased out of all proportion to the temperature.

The symptoms of concealed hemorrhage will largely depend upon the rapidity of blood loss. If the blood loss is continuous but slight the transition from a condition of comparative well-being to that showing a typical picture of hemorrhage will be by such easy stages as to deceive the observer unless he has in mind the possibility of bleeding. Of outstanding importance is the gradual continuous and progressive increase in the frequency of the pulse. The pulse in hemorrhage is quick, shows less volume and there is a peculiar snap as each pulse beat impinges upon the examining finger. The respirations are invariably shallow and frequent, with a suggestion of air hunger, although I have seldom seen marked air hunger. The skin is moist, owing to a lack of vessel tone. There is a distinct change in the color of the patient. The patient is not only pallid but there is a pearly-like tint to the sclera, the facies has

an expression of acuteness and alertness: there are purposeless movements of the arms and legs but as a rule not of the body. The patient subconsciously restrains body movement in the desire to obtain the greatest amplitude of inspiratory respiration. There is a distinct loss of surface heat and the patient complains of an inexpressible sense of weakness.

The first and most outstanding question to be answered: is the hemorrhage continuing or is it stopped? Are we dealing with a single massive hemorrhage, dangerous to be sure, but arrested, or are we having a continuous and continuing hemorrhage? The type of operation and the pathology exposed at the operation very often will help in the consideration of this question. The bleeding from a gastroenterostomy is apt to be interrupted and repetitive, while bleeding from a single large vessel, such as the broad ligament, is apt to be continuous and a lethal termination occur within a relatively short time.

The essential element in the treatment of any hemorrhage is to stop the bleeding and the best time and place to stop bleeding is at the time of the operation. No patient should be returned to bed while the possibility of hemorrhage is still present. The question whether transfusion should be employed and when, is a question requiring nicety of judgment. To give a patient who has been almost bled white a transfusion is ofttimes the cause of fresh bleeding, with a sudden collapse and death. If the patient suspected of having concealed hemorrhage and in whom the diagnosis is reasonably sure has a pulse that is well maintained although rapid, it is many times wiser to watch the patient and allow the bleeding to be more fully arrested than to give an immediate transfusion. At the end of eight to ten hours give a small transfusion and two days later make up for the blood loss by a larger transfusion.

The loss of blood into the abdominal cavity represents not only a loss of the cellular elements of the blood but a great loss in the fluid volume and since the heart requires a certain periphery resistance to maintain circulation fluid should be administered to the patient by practically all avenues, skin, mouth, vein or rectum, unless there are local reasons against the employment of one or the other. In surgery in the upper abdomen, or of the lower abdomen not involving the colon, proctoclysis of tap water with 10 per cent glucose should be instituted. If the operation has been pelvic, water by mouth in the absence of vomiting should be given and hypodermoclysis in all cases. The intravenous introduction of normal saline is associated with some of the dangers outlined here for transfusion, but not all. If however, the patient is in extremus from actual blood loss transfusion must be done immediately as there is a point beyond which patients cannot bleed without dying and the risk of activating the hemorrhage from transfusion must be accepted in order to keep the patient alive and to allow reopening of the abdomen for hemostasis.

If the surgery has been in the nature of gastric resection or a gastroenterostomy the bleeding will be into the stomach and the patient will vomit a greater or lesser quantity of blood sometime after operation. When surgery has been performed on the large bowel the presence of blood in the bowel lumen below the point of anastomosis will invariably stimulate bowel peristalsis with the evacuation of a fluid stool or a soft stool of fresh blood or partially oxidized dark blood. The bleeding that occurs into the stomach after a gastroenterostomy or resection very soon establishes a vicious circle. The more bleeding that occurs the greater the distention of the stomach and the greater the distention the more apt is bleeding to continue. The introduction of a stomach tube through the mouth or a Levine tube through the nose and the evacuation of all of the blood from the stomach is one of the best means of stopping gastric hemorrhage for it allows the stomach to contract and by contraction lessens the liability of further bleeding. We have practically never seen

harm result from a carefully introduced stomach tube, even as recently as two hours after a gastric operation, provided the stomach contents are aspirated and not removed by lavage.

The elevation of the foot of the bed has a decidely beneficial effect in keeping the diminished blood volume in circulation and by providing more adequate blood supply to the already anemic brain centers. Morphine in sufficient doses to quiet the patient and render the patient less acute is indicated. The application of external heat is highly desirable but great care must be exercised in the prevention of burns. The normal patient may tolerate a hot water bottle applied to the body with impunity but which if applied to a patient suffering from hemorrhage a lesser degree of heat is sufficient to produce extensive burns.

The decision to reoperate a patient suffering from concealed hemorrhage is always a difficult one; the natural hope of most surgeons is that the bleeding has stopped and if arrested nursing and adequate postoperative care will safeguard the patient. There is a time, however, when difficult as the decision is it must be made, as failure to reoperate for concealed hemorrhage before it is too late is tantamount to robbing the patient of any expectation of being saved.

At the present time postoperative concealed hemorrhage is fortunately so infrequent that this decision comes but rarely even in an extensive surgical practice, but it does occur and it is our observation that the decision to reoperate is many times too long delayed. If the decision to reoperate is made an immediate transfusion should be given and the moment the transfusion is terminated, the wound should be reopened and the field of the previous operative procedures carefully canvassed. The technical procedure of operating in a field infiltrated with blood, and one that a short time before has been operated upon, is no easy matter. Here again the type of surgery suggests the most probable cause of the bleeding: bleeding after cholecystectomy is obviously either from an incompletely ligated or a loosened suture on the cystic artery, for bleeding from the bed of the gall bladder is ordinarily not of sufficient magnitude to require reoperation. Packing around an untied cystic artery is not apt to be successful as the control of hemorrhage from this vessel always requires ligation. If a gastroentersotomy suture line is suspected of bleeding, or the site of anastomosis between stomach and intestine, an anterior gastrotomy with partial evisceration of the inside of the stomach is the easiest and quickest way to apply direct ligation to the bleeding point and is satisfactory, while the taking down of an anastomosis will seldom reveal the actual bleeding point and will require every detail of the original operation to be reenacted.

Bleeding from soft tissue beds, paranchymatous bleeding or hemorrhage from separated adhesions, requires packing. The operative field is carefully quarantined by placing sheets of sterile rubber tissue about the area and into this barricade, so to speak, sufficient sterile gauze is introduced to maintain adequate pressure for hemostasis.

After reoperation for hemorrhage all of the measures of combating shock and blood loss should be carried out. As long as the pulse is regular, although very fast, the surgical policy should be: rest, fluid replacement and no stimulation. There is a period of a few hours after operation when a wise and masterly inactivity, with a therapeutic courage to just maintain the patient, is far better than unwise measures aimed at stimulating the patient.

PREVENTION OF ABDOMINAL ADHESIONS*

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THE prevention of postoperative adhesions depends entirely on the prevention of inflammatory reaction and in cases in which denuded surfaces are produced by the operative procedures, or in which preexisting adhesions are separated during operation, on the peritonealization of all such surfaces. If, in spite of every care, postoperative adhesions seem to be inevitable, then every effort should be made to adjust the intestines in such a way that these adhesions will not result in ileus.

Anything which bruises, abrades or irritates peritoneal surfaces predisposes to sufficient inflammatory reaction to produce adhesions. All rough handling, therefore, the catching of peritoneal surfaces with tissue forceps or clamps, or the use of irritating antiseptics like tincture of iodine, should be avoided. Dry sponges and wipes readily produce such injury and for that reason should never be used in the peritoneal cavity, but only warm moist sponges. Moist sponges are also greatly superior to dry in absorbing blood and other fluids; the advantage of such moisture was emphasized by earlier writers who spoke of such sponges as greedy; but this advantage has been largely lost sight of by modern operators. Moistening the wipes and sponges with normal salt solution instead of ordinary sterile water may perhaps render them less irritating, but that has not been demonstrated and the difference could certainly be but slight. (Some ambitious student or interne might perhaps make a study of this by animal experimentation and thus produce an interesting thesis.)

Any foreign matter in contact with peritoneal surfaces, like a cigarette drain or rubber tube, necessarily produces a degree of trauma which predisposes to adhesions, and if left long in contact will infallibly produce them; hence when it is at all possible such drainage should be protected by being placed in contact with some fixed portion of the bowel, as the colon, or by the judicious interposition of the omentum, supported as necessary by a few stitches. This applies particularly to drainage of the gall bladder stump, which can be secured by pulling up the omentum and tucking it in between the liver and the transverse colon, fixing it in place by a few catgut sutures.

Drainage in an appendix operation is frequently necessary, but should almost invariably be secured through a stab opening far over to the right, and with the drainage itself so adjusted if possible, *by the fingers still on the inside of the abdomen*, that the omentum and cecum are utilized to prevent contact with the small intestines. The main incision can then usually be closed completely.

If on opening the abdomen preexisting adhesions are found, these should not be separated unnecessarily if they are so placed and of such a character as to offer no obstruction to peristalsis. Only such adhesions should be separated as are necessary in order to remove the pathology for which the operation is instituted. After the operation is completed every effort should be made to cover over all raw surfaces. This can frequently be done, if necessary, by rolling the affected loop of bowel upon its axis so that the raw surface is brought into contact with its own mesentery. A few stitches can then be inserted to hold it in position and thus there will be no raw surfaces exposed. Sometimes adjacent loops of bowel, each with a raw surface, can be adjusted to each other so as to safely bring the raw surfaces in contact, but care must be

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exercised that there is no space left underneath this union for the entrance of a free loop of bowel which would thus be liable to strangulation. The possibility of using the omentum in all these maneuvers should not be lost sight of, since it seems to have been developed for just such emergencies. Sutures should be of very fine silk or of fine plain or chromicized catgut, and should be inserted with great care and with the avoidance of all unnecessary exposure. It is of prime importance to avoid all tension.

The greatest liability to adhesions, however, occurs in the pelvis after operations for old inflammatory troubles which have resulted in extensive adhesions the separation of which may leave the entire surface of the true pelvis denuded. If nothing is done to protect this the omentum and small bowels will drop in and will inevitably form adhesions with resulting liability to postoperative ileus or other serious complication. This raw surface can always be completely peritonealized by making a free opening into the vault of the vagina at the completion of the operation, or leaving the upper end of the vagina open if the womb has been removed, and passing into the vagina the ends of two or three strips, according to the needs of the case, of washed iodoform gauze, the rest of the strips being lightly placed in the pelvis as a fluff. The sigmoid is then swung around over this fluff and attached by continuous suture to the edge of the peritoneum at the brim of the pelvis. Good judgment should be used in bringing this around to see that there are no angulations produced as the stitches are inserted. In very rare cases the sigmoid is not quite sufficient, and then the space on the right can be filled by mobilization of the cecum. The omentum is then smoothly placed over the mobilized bowel and the incision closed. The gauze fluff should be removed after the lapse of one week, by catching the ends of the strips in the vagina. By that time their removal is almost or quite painless and if the opening has been made of proper size, there is no need of any replacement of the gauze. Vaginal douches should be used after removal of the fluff as needed for cleanliness. The sigmoid soon sinks down into the pelvis and thus makes a complete floor. There has been no raw surface exposed at any time.

Great care should be exercised in closing the abdominal incision to see that the edges of the peritoneum do not come in contact with the underlying intestines or omentum. The peritoneum should be closed by a running catgut stitch, with the edges carefully turned out so that such contact can be absolutely avoided.

POSTOPERATIVE INTESTINAL OBSTRUCTION*

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THE causes of intestinal obstruction in the non-operated abdomen are few. It is true that malignancies (principally carcinomas) of the intestine are apt to occur toward late middle life and early senescence, and that congenital anomalies, volvulus and intussusception must be considered in the diagnosis of intestinal obstruction in early life. During the major part of adult life, however, obstruction of the intestine is a rare occurrence except in these persons who have been subjected to an abdominal operation. Before the days of abdominal surgery, strangulated herniae occupied a similar position of importance as a cause of intestinal obstruction in adult life. In backward communities, where reducible herniae are neglected, operations for strangulated herniae are very frequent. Mission surgeons returning from Africa report a surprisingly high incidence of herniae among the natives, most of which sooner or later become strangulated.

In 88 cases of the authors, of intestinal obstruction during the last few years, in which operation was necessary, 32 cases had had some surgical interference in the abdomen. In this paper, however, the occurrence of intestinal obstruction will be considered as a postoperative complication, and only those cases discussed where the obstruction occurred during the patient's stay in the hospital, or happening a very short time after the patient's discharge from the hospital.

The cause of intestinal obstruction (postoperative) is the formation of adhesions either in the form of "fiddle-strings," broad bands, or the plastering together of loops of intestine with angulations or twists. The final cause of adhesions is obscure and their prevention even more elusive. No one has been able to explain why one simple clean case should be followed by adhesions that obstruct, and another case where extensive manipulations are necessary remain free from symptoms. The plea of "gentleness in handling tissues" should always be made, but it is by no means the whole story. Case 11 came to operation with a three liter multilocular ovarian cyst. With the patient in the Trendelenburg position a low, midline incision was made, and the cyst found immediately below. A trochar was inserted and enough fluid withdrawn to allow the cyst to be delivered from the abdomen. There were no adhesions between the cyst and adjacent organs. The intestines, from the patient's position, were well up in the abdomen and no packing or handling was required. The cyst pedicle was clamped, ligated, sutured, peritonealized without the slightest difficulty, leaving a single small suture line, but to this later became attached the sigmoid colon so firmly in five days that obstruction developed which required surgical relief. It is difficult to imagine an operation on the abdomen where there was less tissue manipulation, and yet how many more serious cases escape this complication.

Another interesting, but most distressing group is the so-called plastic peritonitis case. Here extensive adhesions form between adjacent loops of intestines along varying areas of their contiguous surfaces. This condition is clearly beyond the control of the surgeon, but yet he is faced with the burden of repeated operations. One such case has been operated on four times. In another such case the filmy, sticky adhesions could actually be seen to form on the operating table. This patient developed a second obstruction within a few months, which required operative relief. No chemical or other treatment of value has been found to treat this condition.

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Considered as a postoperative complication it is first necessary to differentiate intestinal obstruction from paralytic ileus. Both conditions are apt to be associated with abdominal distention, tympany and vomiting. An absolute distinction cannot be made because paralytic ileus implies a functional obstruction. But distinction must be made between cases that are to be treated as outlined below for (a) ileus and (b) intestinal obstruction.

As a general rule the later onset of ileus can be predicted at the time of operation. Where abdominal infections have been stirred up, or where there has been undue handling of abdominal organs or intestines, one may confidently expect (and is seldom disappointed) paralysis of the intestine which lasts a varying period of time. Clinically, the patient shows increasing abdominal distention commencing twelve to twenty-four hours after the operation. The abdomen becomes tympanitic and tense. The abdominal muscles feel rigid, but this apparent rigidity is really due to the intra-abdominal pressure. No visible peristaltic waves are seen, and with the stethoscope no borborygmus is heard. Auscultation of the abdomen with the stethoscope is an extremely important part of an examination but is frequently omitted. As the distention increases the patient becomes dyspneic from interference with the movement of the diaphragm. The patient becomes cyanotic from cardiac embarrassment. There is usually a cold, clammy perspiration and the patient has an expression of anxiety and apprehension which may well be reflected in the faces of his attendants. In a short period of time emesis of a small quantity of dark fluid may occur. This is usually the indication for the passage of a stomach tube, which reveals the fact that a large amount of dark, foul-smelling intestinal contents distend the stomach.

It has been noted frequently by the senior author that in these cases the general appearance of the patient (aside from the abdominal findings) bears a close resemblance to that of toxemia from infections of various anaerobic organisms. It has been shown that numerous organisms of the anaerobic group are to be found in the average normal individual. The rapid multiplication of these organisms in a stagnant intestine vitiated by a depressed circulation, would not be surprising, nor is it unlikely that the toxic products of the growth of these organisms has a profound influence on the general condition of the patient.

The clinical picture of a typical postoperative intestinal obstruction is shown in Case III. After a period of uneventful convalescence which may be as short as four days, the patient may suddenly complain of severe, agonizing abdominal pain. The onset of pain may be of startling abruptness. In Case V, the pain came suddenly enough to interrupt a sentence and clip a word in two. Such severe pain is usually of short duration, giving way to a general soreness in the upper abdomen or about the incision, which in a few minutes reaches a crescendo of acute pain. Paroxysms of pain follow one another with definite regularity. Immediately preceding the acute period of pain, peristaltic waves are to be seen in some cases, and even during the quiescent periods, a stethoscope on the abdomen reveals the boiling within. Very soon after the onset of the pain the patient vomits, first the gastric contents and then green, bile-stained, mucus. At first there is usually no abdominal distention. These symptoms are definitely indicative of small intestinal obstruction and immediate treatment is imperative.

Postoperative obstructions involving the colon, and especially the sigmoid as in Case II, are marked by less startling symptoms. Pain may be a less prominent symptom and its paroxysmal character only elicited after careful questioning. Even if this pain is overlooked suspicion of an obstruction must be aroused by the interruption of the fecal stream. Perhaps no other fact alone illustrates as clearly

the need of care of the toilet of the colon in postoperative cases. Vomiting is always a late symptom in obstruction of the colon. Distention, gradually increasing in extent is the rule, but associated with it is an active peristalsis which sometimes may be seen but always may be heard with the stethoscope.

If these symptoms supervene on about the ninth or tenth postoperative day one must be suspicious of a fecal impaction. Each postoperative case should be given an enema every day unless a satisfactory spontaneous evacuation has occurred: in spite of such a "standing order" it occasionally happens that fecal matter is allowed to accumulate and become inspissated, particularly if the intestinal activity has been slowed by morphine. This condition can be ascertained from the clinical notes and occasionally the impaction can be felt through the rectum. The treatment is obvious. This never occurs if the funnel drip is used.

In the treatment of paralytic ileus and intestinal obstruction it is necessary first of all to detoxicate the patient. This can best be done by intravenous injections of 10 per cent glucose. If there has been considerable vomiting the glucose is given in normal salt solution, irrespective of whether there is a decrease of the blood chloride. A liter of this solution is given twice a day. Intramuscular injections of glucose should not be given in a concentration of over 5 per cent. We very rarely give glucose by this method.

Patients showing abdominal distention due to a paralysis of the intestine are further treated by large hot abdominal compresses and almost continuous flushing of the colon with normal salt solution. Between the frequently repeated colon flushings the funnel drip remains in operation. Enemas of milk and molasses are occasionally given, and to be efficaceous these should be of 1000 to 1500 c.c. volume. Injections of surgical pituitrin (1 c.c.) are occasionally given, but the use of drug as a routine is not to be recommended. If the slightest suspicion exists of the presence of an organic intestinal obstruction the use of pituitrin is absolutely contraindicated. Gastric lavage should be practiced early before the patient begins to vomit. The removal of gastric contents gives great relief, and the stomach should be kept empty. This may be done by passing a duodenal tube which is left in the stomach and through which irrigations with normal salt are made every thirty minutes. In some cases we have employed the Cornell continuous suction. It is rarely necessary to subject the patient to jejunostomy for the relief of this condition. The incision and drainage of loops of lower small intestine serves to collapse only the loop that is drained, but as a therapeutic measure is useless.

With the sudden postoperative appearance of symptoms of intestinal obstruction immediate operation is indicated. Case v was operated on two hours after the onset of symptoms. Morphine should not be given until operation has been offered and accepted by the patient. In recent postoperative cases the best approach is through the previous incision. If the previous abdominal wound is completely healed, the incision should be made about an inch to one side. The site of obstruction is usually felt without difficulty, and must be relieved. It is surprising how firmly organized adhesions may become in a very few days. The technic of freeing the obstructive condition must vary with the operative findings. Fiddle-string adhesions are cut and the points of attachment buried with a purse-string suture. Broad adhesions are divided with sharp dissection and an endeavor made to cover raw surfaces.

After obstruction of the small intestine has supervened for some hours it is our practice to insert into the dilated loop of jejunum a catheter which is carried out through the abdominal incision. Irrigations with normal salt can be accomplished through this channel. Although "milking out the dilated intestine" has been advised by many surgeons, we have felt that this

treatment involves too much manipulation for safety.

In obstructions of the large bowel drainage of the small intestine is also accomplished by jejunostomy. In cases of long standing obstruction with marked toxemia, where an attempt to free the obstructing adhesions seems hazardous on account of the condition of the patient, a jejunostomy should be performed through a small high left rectus incision. This procedure may carry a patient out of the acutely toxic period and allow the obstruction to be relieved with safety at a later date. It is frequently noted that after the small intestine has been drained, and the intra-abdominal pressure reduced, as by an ileostomy, jejunostomy, or cecostomy, an apparently complete bowel obstruction will allow fluid and gas to pass.

We consider that the treatment of the conditions discussed above is definite and positive. In abdominal distention (due to ileus or peritonitis) without obstruction, gastric lavage, compresses to the abdomen, colon irrigation and intravenous fluid medication are instituted. If from the increasing pressure, fluid keeps flowing into the stomach, and gastric lavage proves unsatisfactory, jejunostomy is indicated. If the stomach can be kept properly emptied, jejunostomy gives no added advantage. The stomach tube must be passed early.

In intestinal obstruction operation, with or without drainage of the intestine, is indicated as early as possible. Conservative treatment, "Waiting for a kink to unwind" is dangerous, and is usually based upon a lack of careful consideration of clinical symptoms.

The following cases typical of postoperative obstruction, developing soon after operation, have been selected as illustrations:

Case I. Operation, St. Luke's Hospital, January 19, 1924, cholecystectomy. Discharged from the hospital on February 2, 1924. Readmitted on February 15, with history of repeated vomiting, and evidence of first partial and later complete upper intestinal obstruction. Operation February 16 showed duodenum closely adherent to the lower surface of the liver and the gall bladder fossa, which caused complete obstruction of the duodenum. This adhesion was not separated, but a posterior gastroenterostomy performed. Convalescence uneventful.

Case II. Patient aged sixty-nine. Operation St. Luke's Hospital November 1, 1927. Low midline incision. Very large multilocular cyst presented, partly aspirated and delivered with long pedicle arising from left broad ligament. This was tied off and raw surfaces covered. November 4 the abdomen was moderately distended with gas, but fecal matter was returned by colon irrigation. November 5 colon flushes returned practically clear; some gas was passing but very little. The distention was increasing. Patient was nauseated and vomited a small amount of mucus. One thousand c.c. 10 per cent glucose in saline intravenous administered. November 6, 2:30 A.M. patient was having cramp-like pains. Distension still present, and peristaltic waves were seen passing across the abdomen. Occasional gas bubbles passed through the funnel drip. Operation November 6, 1927, 10 A.M. through old abdominal incision. Intestines were greatly distended, both large and small, but free from evidence of peritonitis. Small amount of serosanguineous fluid in the belly. In the left broad ligament the sigmoid was attached with organizing adhesions through a distance of about 2½ inches. When these adhesions were freed, sudden gurgling of gas was heard, indicating that the obstruction had been relieved. A catheter was sewn into a dilated loop of jejunum, drawn out through a gap in the omentum through the upper end of the wound, to make certain of upper intestinal drainage, should the sigmoid again close. Abdomen closed. During the next eight or nine days the patient continued to be moderately distended. Gas and fecal matter were passed. Intravenous injections of 1000 c.c. of 10 per cent glucose in saline were made twice daily for eight days, and once daily for the next seven days. Massive milk and molasses enemas were given frequently and produced the best results. This procedure is dirty but the most effective to iron out kinks and angles in lumen of the bowel. By the fourteenth day the patient was walking and left the hospital without

further complications on December 7. She has been entirely well since that time.

Case III. Operation January 24, 1929. Dante Sanitarium, for two enormous fibroid tumors of the uterus and a cystic right ovary. Supravaginal hysterectomy and right salpingo-oophorectomy. Stump of cervix and right broad ligament peritonealized in the usual manner. Convalescence uneventful. Patient home on the 12th of February, 1929 (home 110 miles from this city). Returned to hospital on February 20 with a history of very severe abdominal pain, coming on suddenly on the evening of February 17. Patient vomited soon after onset of pain. Pain was paroxysmal in character, severe, had continued until her admission to the hospital. Her physician at her home told her she had a "kink which would straighten out." The vomiting became fecal and patient markedly toxic and dehydrated. When admitted to the hospital the patient was in marked acidoses, with deep stupor but could be aroused. Pulse of poor quality, about 100. Temperature subnormal. She was given 1000 c.c. of 10 per cent glucose in salt solution intravenously. Operation under ethylene anesthesia on February 20. Incision through dense clean scar of the old operation. Considerable clear, yellow fluid in the abdomen. Dense adhesions found binding the sigmoid over to the right of the cecum. The terminal ileum was angulated and obstructed in the right iliac fossa. After freeing this obstruction it was found that a small portion of the terminal ileum was bound down in the right iliac fossa by a congenital band of peritoneum which was separated with some difficulty. A catheter sewed in the jejunum was led out through the abdominal wound. Patient received a second glucose in salt intravenous infusion, but expired twelve hours later, a victim of delay and conservatism.

Case IV. Patient was subjected to 2 cesarean operations. During the three or four years following the first cesarean operation the patient had frequent symptoms which were undoubtedly attacks of partial and almost complete intestinal obstruction. These cleared. We were called upon to do a second cesarean operation, but at the time of this operation no exploration was made, since a long period of time had elapsed since the last attack of partial obstruction, and there seemed to be no indication for prolonging the cesarean operation by an abdominal exploration. Two or three days following the cesarean operation the patient developed complete obstruction of the small intestine. This condition called for another abdominal operation. On opening the abdomen it was found that the mesentery of the transverse colon was tightly adherent to the posterior parietal peritoneum and was pulled down before the ligament of Treitz, leaving a small ring through which more than half the small intestine had herniated. This hernia had never become completely occluded until the distention occurred, which followed the insult of the cesarean operation, with its associated postoperative ileus. At the time of operation this hernia was angulated sufficiently to completely block the bowel in this ring.

Case V. Aged twenty-four. Operation St. Luke's Hospital March 13, 1930, for right ovarian cyst and chronic appendix. Low, midline incision. Right ovarian cyst and appendix removed in routine manner without difficulty. Abdomen closed. Convalescence entirely uneventful. Temperature normal. Pulse 80–90. Patient allowed up and around ward on the tenth day. On March 23 at 8:00 P.M. patient had sudden onset of excruciating pain in the right lower quadrant and vomited dinner eaten at 6:00 o'clock. Vomitus continued until operation at 10:00 P.M. Pains definitely paroxysmal and very severe. Abdomen sore between paroxysms which came every two or three minutes. Peristaltic waves seen. Operation March 23 at 10 P.M. through old incision. Two adhesions of the ileum found to the cecum, with a loop herniated through the space between these and also attached. These adhesions were very well organized and were freed with careful knife dissection. Raw surfaces peritonealized. Postoperative convalescence entirely uneventful.

ACUTE DILATATION OF THE STOMACH*

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ACUTE dilatation of the stomach, a rare primary lesion, is seen by far most commonly as a sequel to laparotomy. McIver[1] has shown experimentally that this phenomenon may take place at any time under general anesthesia where there is a possibility of the free passage of air through the esophageal orifice, and this is the probable explanation of its occurrence in the majority of cases where the site of operation is extra-abdominal.

The dilatation itself is of the nature of an acute paralysis of the gastric walls and, though occasionally limited to the stomach alone, it is more frequently accompanied by similar changes in the upper intestinal tract. Whether or not the primary paralysis was caused by air drawn into the stomach during anesthesia, the atonic viscus soon contains secreted, imbibed or regurgitated fluid. Probably the most common cause of the gastric paralysis is infectious material in the abdominal cavity and in these instances the duodenum and jejunum are invariably similarly involved. It will be found, moreover, that a varying degree of gastric dilatation is responsible for, or at least intimately associated with, the stormy convalescence seen, and often otherwise unexplained, in clean operations upon the lower abdominal structures or the bile passages.

In most instances, the diagnosis is not difficult. The blown-up, drum-like abdomen with visible or palpable tumor mass on the left side; the constant effort to bring up gas, sometimes in vain, sometimes with copious and recurrent eructations, often with foreboding hiccough, are signs which should lead any observer to a correct interpretation of the underlying condition. In its earlier stages the process is not so easily determined, as the stomach is not entirely lacking in tone and the most common symptom is frequent effortless vomiting, usually of clear or slightly bile-stained fluid. If, at this time, proper treatment is instituted we shall seldom see the extreme condition first pictured. However, if the condition is allowed to progress, symptoms of toxic absorption from the paralysed stomach and duodenum overshadow those of mechanical origin, and the face rapidly becomes ashen or even cyanotic, the pulse becomes rapid, respirations are rapid and shallow, and shortly before, sometimes just after death, immense quantities of blackish-brown or dull greenish fluid flow from mouth and nostrils. The outflow, which gushes rather than is vomited, is caused by the gas associated with the condition and not by active contraction of the stomach musculature.

Acute dilatation of the stomach, looked upon with so much alarm by early writers, should no longer be counted as a possible cause of death. The suspicion that such a condition exists can be verified by the passing of a duodenal tube and treatment for its relief can be as simply carried out. Gastric dilatation cannot cause death unless, unsuspected, it remains untreated.

Having suspected its presence, a duodenal tube of the Levin[2] type may be slipped through the nostril and passed into the stomach without difficulty. If the tube has been well lubricated with petrolatum, jelly or glycerine, it is unnecessary to cocainize the nose or throat though this is sometimes advisable in a nervous or excitable patient. Five per cent cocain on an applicator is the local anesthetic of choice. By the use of the tube described, the stomach may be kept continuously empty without the oft repeated, distressing gastric lavage through a large stomach tube. Moreover, there is ample evidence to show that the stomach once

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emptied by gastric lavage refills rapidly with regurgitated and secreted fluid unless a continuous mild suction is maintained.

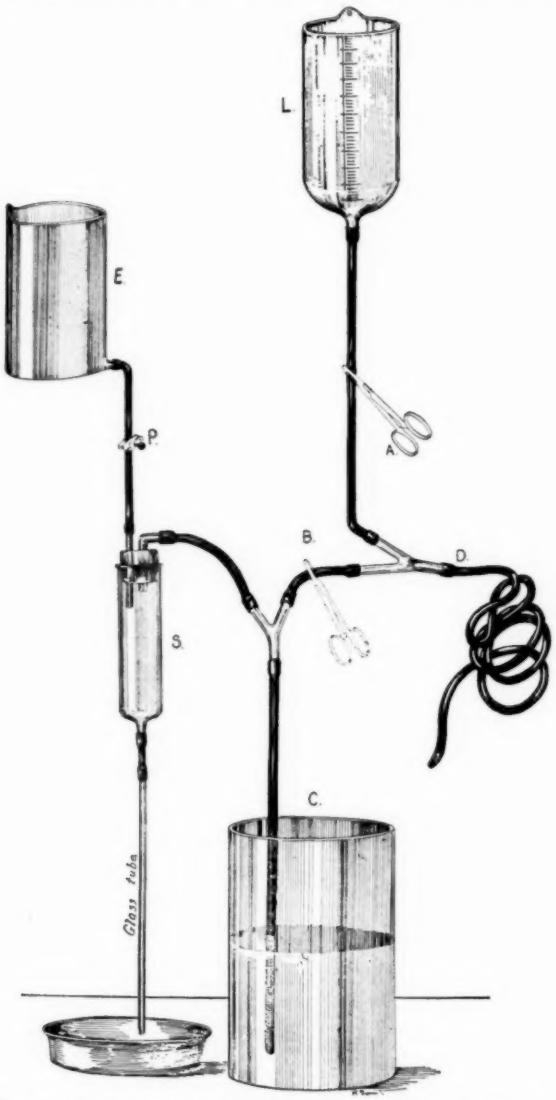

Fig. 1. Apparatus for Continuous Gastric Drainage. D. Levin duodenal tube, No. 14 F., to be slipped through nostril. A, hemostat clamped on tube to lavage solution, L, which is changed to B only during lavage; C, drainage tube, the end of which must be kept submerged; S, Connell suction apparatus, the barrel of a triumph syringe fitted with two-holed rubber stopper. Constant dripping from container, E, regulated to about 100 drops per minute by petcock, P, causes a mild negative pressure due to air bubbles carried down the glass tube between drops of water.

For the application of this suction we have devised an apparatus as illustrated in the accompanying diagram (Fig. 1). This method[3,4] has been used for more than five years in the Surgical and Gynecologic Service of the University of California Hospital and we feel it has saved many lives. It has been used in paralytic ileus, which may or may not be due to peritonitis, in intestinal obstruction, in acute gastric dilatation and even in persistent postoperative vomiting not accompanied by dilatation or ileus. Its manifold advantages, pointed out by Brown[5,6] are: (1) Relief from conditions caused by gas and regurgitated fluids is obtained. (2) There is either interrupted or continuous lavage of the stomach and, in some cases, of the duodenum. (3) Nausea and toxemia are relieved. (4) The patient may drink water freely, relieving that most distressing symptom, thirst. (5) Transgastric feeding and medication are made possible. (6) The patient is so much more comfortable that he often begs for the eturn of the duodenal tube after having once experienced the relief afforded by its use.

When gastric or duodenal drainage is prolonged, care must be taken to prevent alkalosis. Because of long continued drainage, large amounts of chlorides, secreted into the stomach as hydrochloric acid, are lost to the system and alkalosis results. These chlorides must be replaced by massive subcutaneous injections of normal saline solution. In this way the ominous drop in blood chlorides is prevented and the fluid balance of the body is maintained. Normal or 4 per cent sodium chloride solution should replace bicarbonate of soda solution for gastric lavage. Glucose should be administered intravenously for its nutrient value if food by mouth is withheld for long.

SUMMARY

Acute gastric dilatation, formerly considered a serious and frequently fatal complication, should no longer be a possible cause of death. Suspicion of its presence should lead to a speedy test by transnasal insertion of a duodenal tube. Treatment by the apparatus for continuous gastric drainage herein described is simple and rapidly efficacious.

[Note: For references see authors' reprints.]

PREOPERATIVE AND POSTOPERATIVE

THERAPEUTIC USE OF DEXTROSE (GLUCOSE)*

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DEXTROSE or glucose as it is more commonly known, is one of the most important therapeutic agents developed within recent years. The underlying basis for its use is simplicity itself, because "in any diseased state nothing could be much more fundamental as supportive treatment than to supply a patient with food and water in such a form and by such a route that these are immediately available to the starved and thirsty tissues."[1]

During the past five years or more much has been written about the surgical aspects of the use of dextrose. On reading over the literature one is struck immediately by the diversity of ideas expressed and opinions offered regarding certain essential matters such as indications, dosage, good and bad results, and whether or not insulin should be added, to say nothing of the general disregard of how very slowly intravenous injections of dextrose *must* be given in order to obtain full therapeutic effects and to avoid various undesirable consequences.

Probably this is due to the rapidity with which this old-new agent has sprung into its present prominence so that many of us using it still fail to appreciate that a dose of dextrose is a determinable matter definitely governed by rules of rational therapeusis. For example, a recent publication on the intravenous use of dextrose under certain conditions would be of inestimable value except that the author advises its injection by means of a modified blood transfusion outfit, apparently as rapidly as it can be pumped in. Such a method entirely disregards the consequent strain on the renal threshold, the wasteful spill of sugar through the urine, the hyperinsulinism and reactions from overstimulation of the pancreas, possible overloading of the weakened heart muscle, as well as the incontrovertible fact that the therapeutic effect of the dextrose is dissipated and lost in direct ratio to the excessive rate of its injection (or faster than 0.8 gm. per kilo body weight per hour).[2] This same author would say nevertheless that he obtains good effects with his ways and of course some benefits are to be expected, but it may be pointed out that his results would be still better from the same dose properly given, the effects less erratic and uncertain, and the glucose reactions which he does not detail could be entirely avoided by the observance of a proper technic.

In this clinic as probably in many others the combination of obstetrics and gynecology in one service afforded an unusual opportunity for intimate contact and interest in the development of dextrose therapy as a standard treatment for the pregnancy toxemias, only to find suddenly that we had been carried along into an active application of this same agent to surgical gynecology. We use it preoperatively by mouth as a preventive of post-anesthetic nausea, vomiting and acidosis; each major operative case is routinely given at least one intravenous injection of hypertonic dextrose solution as an immediately postoperative procedure; we object, with valid reasons, to the addition of insulin for these non-diabetic patients since we believe that it is not more insulin to burn up available sugar which is required, but rather more sugar to be burned, whilst the patient is perfectly capable of forming in her own pancreas all and more than all the insulin she

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needs; we regulate our dextrose dosage and rate of administration; and we do not have reactions.

It is similarly used in other divisions of this hospital and our services are notably free from such postoperative complications as even ordinary vomiting, acidosis and shock. Moreover, patients thus treated react from the operation more favorably and promptly, do not complain of thirst or nausea, and actually seem to regain even their intestinal peristaltic tone more promptly than in former days.

GENERAL CONSIDERATION OF DEXTROSE THERAPY

A knowledge of certain fundamental facts regarding dextrose metabolism and dextrose therapy is essential in order to obtain uniform results from its use. These can best be discussed in a briefly tabulated form:

Dextrose and Glucose: Originally the word glucose was synonomous in a chemical sense with dextrose. Practically, however, there was a great difference because then, as now, glucose was prepared on a large scale for commercial purposes. Glucose sold as such over the druggist's counter still contains various impurities, and reactions and even deaths have followed its intravenous use.

It was on this account that in the Tenth Revision of the U. S. Pharmacopeia in 1926 need was realized for the establishment of certain fixed standards. At this time the term *dextrose* was officially adopted and listed for that sugar so prepared and purified that it could be used for all therapeutic purposes.

The medical use of the term glucose is no longer correct, therefore, because glucose is the impure commercial sugar or syrup, and one should speak now only of dextrose in referring to the medicinal agent which has so commonly been called glucose.

For intravenous use chemically pure dextrose is no longer necessary if "dextrose, U. S. P. X." is specified.

Reactions following the intravenous use of dextrose solution are unnecessary and avoidable[3] providing no technical errors are allowed in its preparation or its administration. Safe solutions are easily prepared in any hospital, or recourse may be had to one of the many hermetically sealed ampules of dextrose solution now on the market providing they contain no cresol as a preservative. These may be employed without risk of reactions unless the operator fails to use freshly distilled and immediately sterilized water in their dilution, or else gives the solution too rapidly, or too cold, or in overdose.[1,4]

Modes of Administration: Dextrose in solution may be given by mouth, by rectum, subcutaneously or intravenously.

The first two routes probably require little or no comment but certain points common to the latter two need some clarification.

Dextrose solution in itself is not especially irritating when given subcutaneously regardless of its concentration, and when sloughs occur as they occasionally do these are probably the result of pressure necrosis from having been injected too rapidly.

These necrotic sloughs were more common formerly, before the days of intravenous injection, when submammary infusion was the only substantial route of extraoral administration. One probable cause has been alluded to, but it will be remembered that "glucose and soda" was a favorite combination in solution. Bollman[5] has pointed out that alkalies cause a rapid disintegration of glucose or dextrose when heated, so that they should never be added to the sugar solution before sterilization. This and the chemical degeneration of sodium bicarbonate into the more caustic sodium carbonate under sterilization are probably additional factors in the causation of these sloughs.

In any event it is apparent from the foregoing that sodium bicarbonate has no place in combination with dextrose solutions for intravenous injection, and in

fact also should not be used for subcutaneous infusion if such a method of administration of dextrose must be employed.

By the intravenous route underdosage and over-rapid injection are grievously common faults. The consequences of the latter have already been alluded to in an earlier paragraph.

Regarding dosage careful investigations[1] have disclosed that a single therapeutic dose of dextrose for an average sized adult is approximately 75 gm. The maximum rate at which this should be given is 0.8 gm. per kilo body weight per hour[2] in order to have it completely utilized by the body. Translated this means that such an injection of an average therapeutic dose to an average sized adult requires approximately one hundred minutes or *one and three-quarter hours!*

More than this amount at this maximum rate is likely to overstimulate the pancreas in its insulin-producing capacity resulting in an endogenous hyperinsulinism accompanied by hypoglycemia and actual reactions from this source. The same ill result is to be expected even more constantly from the intravenous drip of Matas[6] or venoclysis as it is called by Hendon[7] unless the rate of injection is reduced in these prolonged injections to about one-third that speed at which single therapeutic doses can be given. Hendon even reduces his dosage to one drop per patient's pulse beat.

The fallacy of adding insulin to such injections of dextrose in these normal non-diabetic individuals is emphasized by these important facts.

The strength of the dextrose solution to be chosen depends on various factors such as the condition of the myocardium, the degree of dehydration, acidosis, etc. My own preference for single doses is 300 c.c. of 25 per cent solution, since the effects of the hypertonic solutions seem more prompt and more pronounced. A 15 or 10 per cent solution may be used (500 to 750 c.c. respectively) if more water is required.

For prolonged venoclysis the solution should not be stronger than 5 per cent but this injection may be continued even for days if, as said before, the rate of injection is reduced to one-third the maximum of the Woodyatt and the Wilder and Sansum formula or, not more than 5 or 6 c.c. per minute. Gallie and Harris[8] say that they prefer salt solution for this purpose because dextrose solutions may result in thrombosis of the veins if the injection is prolonged. My own belief is that this objection is largely theoretical while the therapeutic value of the dextrose is greatly superior to that of salt; moreover such injections have been employed in our hospital a vast number of times without any such difficulty.

It is also essential in the prevention of reactions to maintain heat in the solutions during their injection. The administration of cold solutions is probably a frequent cause of reactions.

INDICATIONS FOR DEXTROSE ADMINISTRATION

PREOPERATIVE: In all elective operations the patient should be directed to increase the carbohydrates in his food for a period of several days before his operation. Well sweetened orange juice, or other fruit juices with cane sugar or powdered dextrose should be freely drunk; honey may be eaten with meals; light desserts including ice creams and fruit ices are advisable, and clear hard candy (no chocolates or bonbons) may be eaten after meals.

The patient's carbohydrate reserves will thus be built up in advance; the anesthetic will be less toxic, and postanesthetic nausea and vomiting will be materially lessened by this preliminary preparation.

In such surgical emergencies as traumatic shock, intestinal obstruction, or prolonged preoperative infection as from prostatitis or certain cases of cholecystitis, an intravenous injection of dextrose solution with or without the addition of sodium chloride (depending on the presence or absence of vomiting with loss of chlorides)

should be given while getting ready for operation. In these instances the injection should preferably be made of one of the weaker solutions given fairly rapidly. This supplies the necessary volume of fluid but still need not exceed the formula of Woodyatt, and Wilder and Sansum for the introduction of the sugar.

Many a bad operative risk can be turned into a fairly safe surgical venture by a judicious period of preoperative preparation in which dextrose injections properly chosen and properly carried out play an important rôle.

Postoperative: Postoperative indications for intravenous dextrose administration are three fold: (1) Routine supportive treatment as a restorative measure, or as preventive or active treatment of vomiting and (2) preventive, or active treatment of shock. These are immediate postoperative indications, whereas there is a third group having slightly more remote relationship to the operation. In those cases (3) where peritonitis is a complication as following a ruptured appendix, or peritonitis with profound toxemia as with intestinal obstruction this treatment gives some of the most spectacular results which are to be seen in any class of cases. Postanesthetic pneumonia[9] as well as acute nephritis with urinary suppression also fall into this third group.

Probably the simplest way of considering these conditions and their treatment in detail is to outline them in tabulated form as follows:

Group I. Routine Postoperative Injection as a Restorative, and as a Preventive or Active Treatment of Vomiting and Acidosis:

1. As routine postoperative measure; 300 c.c. of 25 per cent dextrose solution injected intravenously at 3 c.c. per minute. Repeat once or twice if necessary at four to six hour intervals.

2. This is also preventive treatment of postoperative vomiting.

3. If treatment is instituted promptly the same procedure outlined for routine injection should be vigorously pushed for postoperative nausea and vomiting. If vomiting has been excessive before treatment is begun add to solution 1 to 2 per cent sodium chloride to compensate for loss of chlorides in emesis.

4. Proctoclysis of 10 per cent dextrose or of tap water by Harris tidal-stand method is preferable to the Murphy drip.

Group II. Preventive or Active Treatment of Shock:

1. After prolonged operation give 400 c.c. 20 per cent solution of dextrose intravenously at 4 c.c. per minute, with intrarectal injection of one pint of hot dextrose (10 per cent) with sodium chloride (2 per cent). Repeat intravenous injection once or twice at from four to six hour intervals if necessary.

2. Active treatment of postoperative shock; 750 c.c. of 10 per cent dextrose solution intravenously at 7.5 c.c. per minute, or if more haste is necessary 750 to 1000 c.c. of 5 per cent dextrose solution with 1 per cent sodium chloride at 15 c.c. per minute; or 500 c.c. gum acacia and dextrose solution at about the same rate.

Valuable time should not be wasted on attempts at venopuncture. The veins are frequently so collapsed as to be difficult of access by needle and should be cut down upon without delay. The observance of the technic of Larkin,[10] of lifting a triangular flap in the vein, and then closing the wound with an encircling suture tied in a bowknot after the injection is ended makes it possible to use this same vein anumber of times for subsequent injections if necessary.

Group III. Suspected or Actual Peritonitis, Intestinal Atony or Beginning Ileus, and Toxemia Following Intestinal Obstruction.

1. With suspected or local peritonitis give 300 c.c. of 25 per cent dextrose solution intravenously at 3 c.c. per minute repeated after four to six hours for day or two. With general peritonitis of mild degree this same procedure will be sufficient, and this is also true of intestinal atony or beginning ileus.

2. If these conditions have existed for some time or are of serious degree and do not respond quickly and sufficiently, change to prolonged intravenous administration (venoclysis) giving 5 per cent dextrose solution through tied-in cannula at about 5 c.c. per minute. Continue this over period of several days without interruption. In cases of marked peritonitis where vomiting has been severe, and especially after operation for intestinal obstruction from 1 to 3 per cent sodium chloride should be added, until blood chlorides are restored to normal.

3. Following operations for intestinal obstruction venoclysis of 5 per cent dextrose solution with or without 1 to 3 per cent sodium chloride is not merely recommended but urged as a routine procedure. This should be continued as long as five or six days if necessary.

It is, of course, understood that the foregoing is merely an important addition to other rational methods of treatment already established, and it is probably unnecessary to point out that there are many other surgical indications for the use of dextrose, particularly by the intravenous route. The occasions for its use will constantly suggest themselves to the surgeon who keeps in mind the fact that food and drink are essential to any individual, and that to furnish such elemental but necessary support to a patient who is seriously sick may frequently be the determining factor between his recovery and his death.

SUMMARY

1. The preoperative and postoperative administration of dextrose (D glucose) is now regarded as of great importance in the general care of the surgical patient. Intravenously injected dextrose will frequently convert the bad surgical risk into a comparatively safe surgical venture.

2. Administered preoperatively by mouth or by vein as necessity may require it acts as a preventive of postanesthetic nausea, vomiting and acidosis.

3. Routine postoperative intravenous injection of hypertonic dextrose solution is recommended in the prevention of vomiting, acidosis and shock, intestinal atony, etc.

4. Dextrose solution injected intravenously is effective supportive treatment for traumatic shock, peritonitis and the toxemia of intestinal obstruction.

5. Modes of administration, dosage and indications for dextrose therapy are discussed in this paper.

6. The assertion is again made that unfavorable reactions from intravenous injections of dextrose are avoidable if certain technical but simple essentials are understood and observed by the operator.

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POSTOPERATIVE SALIVARY GLAND INFECTIONS*

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AMONG the complications which may ensue after surgical endeavor, acute salivary gland infections rank low in incidence but high in actual and potential danger. Whether or not its occurrence is an indication of low resistance, whatever its etiology may be, its significance is always sinister.

Much has been written on this topic, notably the parotid gland involvement, because it is the most common, far exceeding submaxillary and sublingual in frequency. A review of the literature by the writer in 1923, disclosed contributions by surgeons of many countries, from Paget in the eighties of the last century down to our time. These surveys indicated no unanimity of opinion as to etiology, but did point out its seriousness, all concurring that the average mortality was about 30 per cent. The accurate computation of mortality is difficult, because the condition so frequently manifests itself as a complicating factor in a patient already gravely sick.

The literature since 1923 has been similarly reviewed. A number of writers have contributed scholarly studies of this most distressing lesion. They, however, have shed no light on the causative factor. Speculation continues as to whether it is hematogenous, ascending (via salivary ducts), traumatic (by anesthetist), or lymphatic. The preponderance of evidence leans to the first two. It is pointed out that trauma in general, in industry, in boxing and other rough sports, is not followed by parotitis. As for lymphatic approach, the parotid lymph nodes derive lymph only from the gland itself, the external ear and eyelids, which makes it doubtful that the infection reaches the gland through lymphatic channels.

There is much evidence to support both the hematogenous and ascending etiologic concepts and it may well be in many instances that a combination of both factors enters into its genesis. The adherents of the ascending or ductal theory base their arguments on a dry mouth, salivary stasis and the fact that cultures from the gland, Stenson's duct and the mouth are identical in postoperative parotitis. Complementing this is the observation that the inflammatory process begins in the center of each lobule. Resemblance to ascending biliary, urological and mammary infections is also advanced in support of this theory.

However, postoperative patients generally suffer from dry mouths and some degree of salivary stasis, if not oral sepsis. If these factors are potent, salivary infections should be more frequent. The fact that the invasion is so generally of the parotids is explained on the hypothesis that saliva produced by the sublingual and submaxillary glands is more bactericidal than that of the parotid and also that the ducts of the former open under and are kept clean by movements of the tongue. Stenson's duct opens outside the alveolar process, near the molar teeth, which are frequently carious.

The rapid onset after operation, the extent of the involvement and the associated septic symptoms, on the other hand, point to a hematogenous source, entirely apart from those instances, in which the occurrence is manifestly pyemic.

The diagnosis offers no difficulty as a rule. A tense, tender swelling develops in front of either ear. The skin is red. The swelling is painful. There is a rise in temperature, with associated prostration and discomfort. Such is the familiar picture of acute parotitis.

It may slowly subside on the involved

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POSTOPERATIVE SALIVARY GLAND INFECTIONS (ST. CATHERINE'S HOSPITAL)

Disease and Operation	Age	Sex	Nativity	Involvement	Length of Time Post-op. (Days)	Incision	Culture	Hospital Stay (Days)	Other Complications	Result	Diet Post-op.	Mouth Throat
1925 Chronic appendicitis Chronic cholecystitis Appendectomy and cholecystectomy Wound clean	22	M.	U. S.	Left	10	No	None	41	None	Cured	Fluids	Teeth and tonsils good
Ureteral calculus Ureterotomy Wound infected	41	M.	U. S.	Both	8	No	None	34	None	Cured	Fluids	Teeth fair
Incision and drainage Acute appendicitis Acute gen. peritonitis Patient very ill on admission, T. 104°F. Parotid cleared up after 11 days. Patient lived 18 days after.	53	F.	Ireland	Left	25	No	None	54	Pelvic peritonitis General sepsis	Died	Fluids	Teeth poor
1926 Strangulated umbilical hernia Repair, wound infected	58	F.	U. S.	Both	6	No	None	10	Fecal fistula	Died	Fluids	Teeth very poor
Acute appendicitis Acute gen. peritonitis Appendectomy Wound infected	14	M.	U. S.	Left	13	No	None	29	Secondary hemorrhage of wound	Died	Fluids	Teeth good
Acute perforated appendix Acute local peritonitis Appendectomy Wound infected	26	M.	U. S.	Left	3	Yes	None	28	None	Cured	Fluids	Teeth fair
Cholelithiasis Cholecystectomy Wound clean Parotitis subsided after 5 days	58	F.	U. S	Left	3	No	None	20	Secondary hemorrhage from wound	Died	Fluids	Teeth good
Acute appendicitis Incision and drainage for app. abscess Wound infected	69	F.	Italy	Right	6	No	None	12	Pulmonary embolism Diabetes	Died	Fluids	Teeth fair
1928 Perforated duodenal Ulcer Excision and suture Wound clean	49	M.	Russia	Both	4	No	Staph. albus	11	Wound rupture General sepsis	Died	Fluids	Teeth poor
Toxemia of pregnancy Cesarian section Wound clean	30	F.	U. S.	Right	7	Yes	None	27	None	Cured	Fluids	
Intra-abdominal Abscess Incision and drainage Wound infected	63	F.	U. S.	Both	4	No	None	16	None	Died	Fluids	
1929 Inguinal hernia Hernioplasty	10	M.	U. S.	Both	2	No	None	15	None	Cured	Fluids	Teeth poor
Chronic appendicitis Appendectomy Wound infected	16	F.	U. S.	Both	5	Yes	Staph. aureus	27	None	Cured	Fluids	Teeth good

side as it appears on the opposite side. The latter may quiet down and the original side again become active. Suppuration may or may not occur. If it does, spontaneous drainage may eventuate through the external auditory canal, the temporal fossa or the pharynx.

Prophylactic treatment emphasizes the necessity of adequate oral hygiene before and immediately after operation. Careful cleansing of the mouth, keeping the oral mucosa as moist as possible, postponing elective operation in the presence of bad oral hygiene are all important.

Once it develops, iodine and ice locally are useful, chewing gum or acidulous

lozenges (lime or lemon) are helpful in stimulating a flow of saliva. The free intake of fluids is essential and in the bad cases, hypodermoclysis is beneficial.

If pus forms, obviously drainage is necessary. Incision may be made at the level of the angle of the inferior maxilla, keeping in mind the location of the facial nerve. It should preferably be horizontal and once the dense sheath is opened, the gland proper may be entered with a blunt forceps.

Fisher,[1] in a recent study, advocates the daily intravenous use of 5 per cent mercurochrome. He considers it effective, especially in those infections due to staphlyococci, often aborting a diffuse, destructive process. He also, when drainage is needed, describes a Y shaped incision, one arm of which begins at the zygoma, the other at the mastoid, both joining below the angle of the jaw to continue down to the supraclavicular notch. The writer has had no experience with either, but believes they have merit.

A review of the records at St. Catherine's Hospital, for the past five years, discloses 13 cases with 7 deaths. Further analysis of the record removes 4 of the deaths for the reason that the parotitis was not a determining factor. In 1, the parotid infection subsided eighteen days before death, 2 had secondary wound hemorrhages and 1 died of embolism. The mortality in this group, as accurately as it can be estimated, is therefore about 24 per cent.

[1] Fisher, W. H. *Ann. Sur.*, 86: 445–448, 1927.

SUCCESSFUL MOUTH TO MOUTH INSUFFLATION IN THE ASPHYXIATED ADULT

A NEW METHOD*

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NEW YORK

THERE is probably no emergency in operative work which is so demoralizing and fatal as acute asphyxia. The picture of the patient whose respirations are completely obstructed, who passes rapidly from cyanosis to lividity, dilated pupils, and finally, through muscle spasm to the complete relaxation of the antemortem state, is one which we can never forget.

In the face of this condition, what do we ordinarily do to meet it? Put a mouth gag between the teeth, pull out the tongue, do Sylvester artificial respiration, dilate the sphincter, give hypodermic injections, and make other gestures, usually without any result.

In a previous communication,[1] the writer has met this condition by laryngoscopy, suction, intubation and insufflation of oxygen CO_2. This method, which is growing in popularity in the resuscitation of the newborn, has also been found valuable in the adult.

The insufflation of oxygen and CO_2 is of the greatest possible value after laryngoscopy and intubation. Facilities for intubation should always be available for use. A description of the author's pocket flashlight laryngoscope has previously been published[2] and the intratracheal tube employed has been described.[3] In the intervening three years, the writer has come to the conclusion that this equipment is as essential as the anesthetic agent. Its construction is so simple that it does not get out of order.

The following recent case illustrates the extreme value of immediate laryngoscopy and intubation in asphyxia, due to obstruction.

Patient, male, twelve years old. Manhattan Eye & Ear Hospital, N. Y. February 18, 1930. Temperature: 105.6°F. Rather fat, with thick-set, short neck; previously anesthetized on February 9, 1930. The anesthesia at this operation was characterized by extremely rapid respiration, requiring a high percentage of oxygen to prevent cyanosis; air was given frequently without effect, in the hope of reducing the respiratory rate. Recovery had been prompt, without complication.

This anesthesia, the second, took place on February 18, 1930; gas oxygen without ether operation for jugular resection. The characteristic rapid respiration developed within a few minutes with a rate of between 50 or 60 a minute. It was impossible to bring down these respirations by the addition of oxygen or air. The jugular wound had been closed, attention was being directed to the mastoid wound; severe bleeding from sinus had just been controlled. A new incision was made posterially. At this time, the patient's respiration seemed to become obstructed as well as rapid; no pharyngeal tube had been used because of the extremely light anesthesia maintained; his color was cyanotic, and oxygen did not relieve the cyanosis. The neck seemed to be swollen and full. Removing the mask, I found the lower lip caught between the teeth which were tightly clenched. The cyanosis increased in spite of oxygen being offered to the patient. Anticipating an accident, I called for suction and my laryngoscopic apparatus. The draperies were thrown from the face; the lips were livid. The respiratory effort became weaker and weaker, and finally ceased. The pupils dilated and the clenched teeth relaxed. The small laryngoscope was introduced without difficulty. The soft parts of the airway were deep blue and completely relaxed. A quantity of mucus in the hypopharynx was removed and the glottis located. During this time, the surgeon was compressing the chest, but there was no exchange of air and no respiratory movement. The No. 7 intratracheal tube employed for anesthesia was intubated. There was slight tone of the cords which were closed, but there were no active reflexes and no spasm.

[1] FLAGG, P. J. Treatment of asphyxia in the new-born. *J. A. M. A.*, 91: 788, 1928.

[2] FLAGG, P. J. Exposure and illumination of pharynx and larynx, etc. *Arch. Otolaryngol*, 8: 716, 1928.

[3] FLAGG, P. J. Intratracheal inhalation. *Arch. Otaryng.*, 5: 394, 1927.

*Submitted for publication, March 6, 1930.

I immediately blew forcefully into the tube, distending the patient's chest. At the crest of the insufflation, a convulsive inspiratory effort

Fig. 1. Author's laryngoscope showing large, medium and baby specula.

of the patient's chest took place, as if to deepen the effects of my insufflation. Allowing the expiration to escape through the tube, I blew in again. The color improved at once; oxygen was then blown into the tube; the patient seemed about to breathe spontaneously, then did breathe through the tube. I insufflated once more, the patient's respiratory effort complementing my insufflation. The color became brilliant, a sharp laryngeal spasm developed from the presence of the intratracheal tube which was immediately extubated. The patient breathed deeply and forcefully and made convulsive movements of the face and body (anoxemic jactitation in reverse order). The patient was then given a pharyngeal tube and pure oxygen with a little ether. The respiration slowed down, ether was stopped, and gas oxygen continued, the patient recovering consciousness on the table.

The cause of this respiratory failure is thought to be due to continued anoxemia from nitrous oxide and oxygen (this

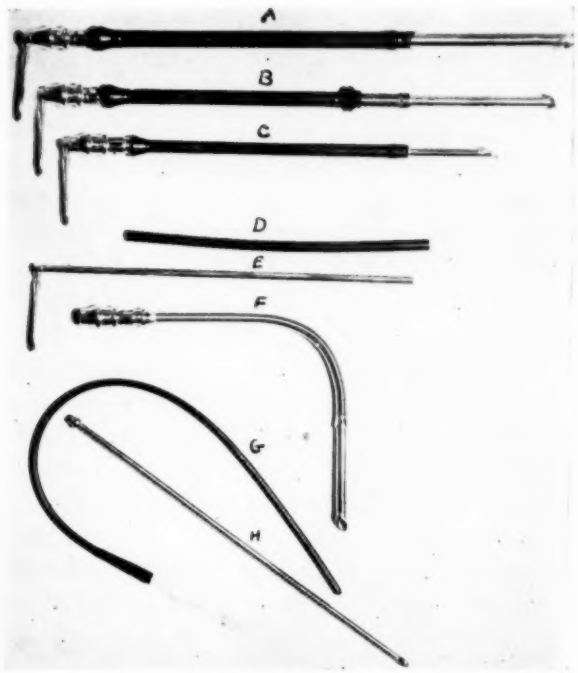

Fig. 2. A, No. 9 intratracheal inhalation tube. B, No. 7 intratracheal inhalation tube. C, No. 5 intratracheal inhalation tube covered with Penrose tubing. D, Penrose tubing. E, Obturator. F, Naked tube with obturator removed. G, Suction catheter of rubber. H, Intratracheal suction tube.

anesthetic having been chosen because of the fact that the patient had an acute nephritis and bronchitis), resulting in complete anoxemia through muscle spasm. The sequence of anoxemia, spasm, cyanosis, relaxation of the masseters and the glottis, dilated pupils, absence of respiratory effort and ineffectual artificial respiration by direct compression would certainly have resulted in death.

The curious phenomena of an inspiratory effort on the patient's part, at the crest of my direct mouth insufflation, rapid oxygenation, forceful expirations, rapid return of laryngeal reflexes upon oxygenation, within 6 or 8 breaths, and anoxemic convulsions during recovery, all tend to confirm the condition as due to oxygen deprivation.

This patient's life was undoubtedly saved by prompt laryngoscopy, intubation and mouth insufflation.

POSTOPERATIVE KIDNEY COMPLICATIONS*

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KIDNEY complications are among the relatively rare but clinically very important sequelae of operations. The following 4 basic forms may be differentiated: (1) Disturbances in the function of the kidney, (2) inflammatory, non-infectious diseases, (3) infectious diseases of the kidney, and (4) the effects of changes in the efferent urinary passages upon the kidney.

Postoperative disturbances of the function of the kidney, which in the majority of the cases are transient but, under certain conditions, may also lead to the death of the patient, may be produced by inhibitory reflexes which originate in the other kidney or in the bladder, when these organs are operated upon. Under such conditions there results either a reflex angiospasm, or a sudden overloading of function through the removal of the relatively still well-functioning sister organ (tuberculosis, hypernephroma), or, finally, the various non-specific insults, which the operation as such exerts upon the organism. The importance of this latter factor, German surgery has learned to appreciate more and more, since it has been shown that some of the cases of postoperative fatality, which are usually attributed to the shock of the operation, are merely masked uremias; for this reason a kidney function test is always done before every major operation, especially laparotomy, in many of the surgical clinics.

Postoperative, inflammatory, non-infectious diseases of the kidney in the form of simple degeneration, nephrosis, or even nephritis are usually attributed to the anesthesia, but in general it may be said that the effect of the anesthesia, especially since chloroform is no longer being used, is markedly overestimated. Braun (Zwickau), who deserves the greatest credit for the introduction of local anesthesia into Germany, repeatedly emphasizes the fact that operations on the kidneys are the very operative procedures that are best done under general anesthesia. It is certainly correct, that entirely intact organs are no longer subjected to the previously mentioned reflex as well as anesthetic injuries, but it must be emphasized repeatedly, that even predispositions are sufficient in this respect, which we cannot discover even with our best functional tests and methods of examination. A certain factor of danger, which cannot be relieved at the time, is inherent in every one of these operations.

The infectious diseases of the kidney, owing to the very frequency of their occurrence, demand by far the greatest amount of consideration. Inasmuch as one of the most important prerequisites of their occurrence are postoperative changes in the efferent urinary passages, particularly in the pelvis of the kidney and ureter, these two latter conditions in this classification may be discussed together.

In the first place, a few remarks on the pathologic-anatomic fundamental principles. There can no longer be any doubt today, that the entire urinary tract reacts to the most varying insults, particularly also to infections, as a unit. This means that urethritis, cystitis, ureteritis, pyelitis, pyelonephritis, abscess of the kidney and pyonephrosis, are merely different localizations, and under certain conditions, different stages of the same disease process. One sees sufficient cases in which the entire urinary tract is actually attacked by the infection. But in contradistinction to such a unitary conception one must not overlook the fact in other cases the localization of the infectious process in a single section of the urinary tract is so predominant, that the conception of the previously mentioned disease pictures as more or less

* Submitted for publication, May 13, 1930.

isolated phenomena is absolutely correct. Inasmuch as the nomenclature in this field is by no means uniform, as a result of which there are basic difficulties in diagnosis, a few definitions of the terms used by us are in place. Under the term "pyelitis," we understand an inflammation of the mucous membrane of the undilated pelvis of the kidney; but if, on the other hand, the pelvis of the kidney is dilated, we speak of an "infected pyelectasis or hydronephrosis." Whereas the simple form of pyelitis always results from an ascent of the infection from the lower urinary passages, hydronephrotic infection may also arise in a metastatic way. A "pyelonephritis" is understood to be a suppurative inflammation of the interstitial tissue of the kidney, whereas the true parenchyma of the kidney is affected only secondarily in the advanced stages. In the fully developed cases one sees infiltrations of leukocytes in the transverse section of the kidney, which pass in the form of stripes along the urinary canaliculi from the cortex to the tips of the pyramids. Abscess formation may occur in any part of this course, most frequently at the surface of the kidney. This disease picture may be ascending, that is, reach the kidney itself from the pelvis of the kidney through an ascending infection, or a descending infection, in which case a metastatic cortical abscess forms at first from bacterial emboli in a glomerulus, from which the infection of the medullary substance of the kidney ensues. In the individual case a differentiation between these two modes of infection is hardly possible.

Formerly the colon bacillus was almost exclusively (up to 90 per cent of the cases) blamed as the exciting germ, but more recent investigations have shown that cocci of a great many types are constantly gaining greater importance (up to 40 per cent of cases) in the hematogenic infections and, particularly, in the sequelae of operative interventions, in which we are especially interested at this moment. In this connection, brief mention is made of the fact that very recently the pathogenicity of the colon bacilli has again been questioned, a view that had previously been presented by Rovsing in 1898, but which had to be considerably modified later.

As is well known, predisposing factors also are always necessary for the occurrence of any infection. In the case of the occurrence of true renal infections, pyelonephritis and abscess of the kidney, these are practically unknown. *The indispensable prerequisite for an infection of the pelvis of the kidney, however, is a motor insufficiency with or without dilatation of the calyx-renal-ureter tube.* To speak more accurately, one would say: a prerequisite for a long-continued infection, as the acute infections of the mucous membrane of the renal pelvis, like every other superficial infection of a mucous membrane, shows a marked tendency toward a spontaneous recovery. A chronic suppuration originating from the renal pelvis, therefore, results only when this spontaneous recovery is prevented by complicating factors, and this happens almost exclusively as a result of a motor insufficiency of the efferent urinary passages and the backward stasis of the urine associated with it. *From this is drawn the clinically exceedingly important conclusion, that the diagnosis of "chronic pyelitis" as an independent disease picture is always a wrong diagnosis!* The chronic suppuration originating in the pelvis of the kidney is never an independent disease, but always merely a symptom, and the aim of the diagnosis is to determine what the factors are that prevent this tendency to spontaneous healing of the inflammation and favor the chronicity of the suppuration. Two pathologic factors enter into the discussion at this point, namely, the dilatations of the renal pelvis mentioned and the infection of the renal tissue itself.

According to the most recent status of investigations, I would like to differentiate 3 types of dilatations of the efferent urinary passages: (1) *congenital atony*, in which

group belong the frequently very large dilatations of the bladder, ureter and renal pelvis in the newborn, but even the hydronephrosis in movable kidneys is, in most of the cases, not a result of the renal dystopy but a phenomenon coördinated with it as a common expression of a universal hypotonia of the entire connective tissue apparatus; (2) *the mechanical dilatation* from peripheral obstruction of urinary outflow, and (3) *the adaptive relaxation*, by which term is to be understood the fact, that with moderate obstruction to the urinary outflow the renal pelvis and ureter reduce the tonus of their walls in adaptation to the changed dynamic conditions and consequently increase their capacity, without the wall being mechanically dilated or even overdistended. This is a very general compensatory mechanism, which we also see in other hollow viscera, for example, in the bladder with hypertrophy of the prostate, in the left heart with aortic stenosis, in the pulmonary alveoli with bronchial spasms, and numerous other conditions.[1] The functional character of these dilatations of the renal pelvis and the ureter and their difference from the definitive atony and dilatation is shown by the fact, that they may retrogress completely and permanently with suitable drainage therapy. The knowledge of this mechanism is of fundamental importance especially in the understanding and the treatment of the pyelitis of pregnancy and of the puerperium. Regardless of the type that has caused the accumulation of residual urine in the pelvis of the kidney, so long as it is present, it presents an absolute hindrance to the cure of the infection, and the first endeavor of every form of therapy consists in relieving the renal pelvis by intermittent or continuous drainage.

An infected residual urine in the pelvis of the kidney, therefore, is the primary cause of the chronicity of a renal pyuria; the second, as has been mentioned, is the pyelonephritis. Whether there is such a condition as an uncomplicated pyelitis, that is, whether or not every infection of the renal pelvis is associated with a more or less extensive infection of the adjacent urinary canaliculi, is an important theoretical question; clinically, both of these conditions, pyelitis and pyelonephritis, can and must be sharply differentiated. In fact, we speak of a clinically important pyelonephritis when the kidney function shows only an indication of diminished function.

The diagnostic procedure in a chronic renal pyuria is, therefore, the following: In the first place, one should determine, with ureteral catheterization and possibly with the aid of pyelography, whether or not there is any residual urine in the pelvis of the kidney. If this is not the case, the chronicity of the pyuria can be due only to a pyelonephritis. In order to confirm this or in order to determine the extent of the destruction of the renal parenchyma, the simplest functional test with indigo carmine or phenolsulphonephthalein is sufficient. A further important differentiation of these two disease conditions is furnished by the clinical symptoms. Without going into any further detail, an almost universally made mistake should be corrected at this time: Pyelitis is never the cause of very severe pains or very high temperatures or even of a chill. *Every urologic disease having its onset with severe clinical symptoms, including high temperatures and chill, is due to either a metastatic infection of a previously formed hydronephrosis or, in the vast majority of cases, to the development of a metastatic cortical abscess in the kidney.* Inasmuch as the infections of the kidney following surgical intervention are almost always of hematogenic-metastatic nature, one may say, *that the embolic pyelonephritis represents the typical postoperative complication as far as the kidneys are concerned.* The sources of the infection are: the infected urinary bladder, from which the kidney may be infected both by an ascending and a

[1] Cf: Schwarz, O. Pathologische Physiologie der Harnblase. In: Handbuch der Urologie. Berlin, Springer.

metastatic route; every pus focus anywhere in the body; and, finally, the intestinal tract.

When we enter upon the discussion of the special pathology of postoperative infectious complications of the kidney, the term "surgical intervention" should be used in the widest possible sense. The simplest, but at the same time perhaps one of the most important of these surgical interventions is catheterization or the introduction of instruments into the urethra and bladder. It is a well-known fact that occasionally these procedures are followed by a chill even with a definitely aseptic procedure. *The cause of this so-called "catheter fever" is always a peracute sepsis, which leads to abscess formation, in rare cases in the prostate, but usually in the cortex of the kidney.* This phenomenon is interesting evidence for the teaching, particularly advanced by the Mayo school, of the organ specificity of germs, in which the germs originating in the lower urinary tract and brought into the blood circulation through the catheterization that slightly injures the mucous membrane of the urethra repeatedly and electively settle in the parenchymatous portions of the urinary tract. Thus, we believe today that with our advanced asepsis such infections are no longer produced by alien bacteria, but by a squeezing of the normal saprophytes of the urethra into the blood circulation, so that in these cases the germs are such as are specifically adapted to the tissue of the urinary tract. The existence of these metastatic cortical abscesses has been proved both clinically and by autopsies, as such catheter infections are usually only transient, but occasionally still lead to chronic or even lethal so-called uroseptic diseases.

It is well known how dangerous the first catheterization of a bladder with much residual urine is. When, in such a case, an infection results, under certain conditions a fulminating sepsis with a rapidly fatal outcome is associated with this apparently very harmless intervention. Usually, however, these infections run a milder course and there results a gradually ascending infection from the bladder to the higher sections of the urinary tract. This, however, never remains confined to the pelvis of the kidney, but always sooner or later attacks the parenchyma of the kidney; there results a pyelonephritis with more or less marked diminution of the renal function and chronic urosepsis. One can almost say, that the purpose of an early prostatectomy is to anticipate these severe complications of the later stages. This ascent of the infection from the bladder to the higher sections is a much discussed but still not entirely solved problem. Aside from the fact that a route of infection via the lymphatics of the ureter has been proved, we know that the prerequisite for such an ascent is an insufficiency of the ureteral musculature including the occlusive mechanism of the ureter, so that the so-called vesiculoureteral reflux occurs. We can well understand that anatomic changes of the ureteral ostium, such as are produced by tuberculosis, spinal disorders, ulcerative cystitis, operative cicatricial distortion and injuries following the passage of stones, lead to a gaping of the ureteral openings. But we are not interested here so much in these conditions, but rather in those cases in which with an anatomically intact ureteral wall the ureter loses its tonus temporarily or permanently. This occurs under anesthesia with the use of local anesthesia of the bladder mucosa with very mild infection and similar harmless conditions, so that even with a slight increase of the intravesical pressure a reflux of the urine as far up as the pelvis of the kidney occurs. The cystographic evidence of these conditions is always easily obtainable. Personally I believe that this phenomenon depends upon a constitutional weakness of the musculature of the ureter and represents the slightest grade of the previously mentioned congenital atony.

We now come to the discussion of the

pathology of the ureter, which represents a very frequent postoperative complication of many types of operations. Gynecological operations, particularly, lead to injuries of the ureter, which almost invariably cause infections of the kidney. Special attention should be called to the fact that these infections of the kidney rob the immediate surgical successful transplantations of the ureters of their permanent results, as these transplantations, even when done with the most perfect technic, can neither replace nor simulate the natural occlusive mechanism of the ureter, so that there results either a wide communication or, more often, there results a stenosis at the site of implantation from secondary cicatricial contraction with subsequent dilatation of the efferent urinary passages, urinary stasis, infection and, finally, destruction of the kidney. Occasional permanent results of such transplantations cannot disprove the knowledge of the best German gynecologists obtained from extensive statistical experience.

Among the postoperative infectious complications in the kidney, all the different operations on the pus foci in the body assume an important position. After Rovsing in 1889 and later German authors, especially, Scheidemantel in 1913, called attention to the significance of tonsillitis, it is to the special credit of Rosenow, Bumpus and Meisser that they emphasized the connection between disease of the tonsils and infection of the kidney and that they considerably extended the discovery of the organ specificity of the bacteria. Here, where the postoperative connections interest us chiefly, attention must be called to the fact that such infections of the kidney result not very rarely during the operation on these pus foci, either because a protective pyogenetic membrane is ruptured by the operation (especially with osteomyelitis), or the bacteria are squeezed into the opened lymph spaces, as happens particularly with tonsillectomy by squeezing and pressure. A not so very inconsiderable number of the acute septic fatalities after such operations have a peracute hematogenic diffuse pyelonephritis as an anatomic substrate.

Finally, the intestine is of the greatest practical importance as a source of the development of infections of the kidney. And as evidence of the importance that we attach to these factors, it should be said from the very beginning that the relative frequency of infections of the kidney in women should not be attributed to the short length of the female urethra, but rather to the relative frequency of the disturbances of motility of the intestines in females. The reports in the literature on the connections observed between infection of the kidney and disturbances in the activity of the gut (especially obstipation) vary considerably. A probable explanation for these differences of opinion might be the following: here, exactly as in the pelvis of the kidney, even a diminution of the motility with such a slight functional disturbance that it is not recognized by the patient, is sufficient and therefore escapes a superficial examination on the part of the physician. Colitis, appendicitis, cholecystitis, and even slight gastrointestinal disturbances lead to obstinate infections of the kidney in spite of all forms of therapy, which rapidly lead to recovery after removal or successful treatment of the primarily diseased portion of the gut.

This relationship between the gut and the urinary tract as a postoperative complication, which is so constant, that the French urologist Heitz-Boyer speaks of an "entero-renal symptoms complex," may become effective when long-continued disturbances of intestinal activity exist in a more or less immediate association with the operation. In this connection, one must also think of the effect of an intervention on the part of the physician which, though not immediately surgical, is very consequential in connection with operations, namely, the use of morphine. There is hardly another therapeutic remedy that exerts

such an unfavorable effect upon all the function of the urinary tract and, among others, in the sense of a predisposition to infections: it paralyzes the secretion of the kidneys, produces hypertonia of the sphincteric musculature of the ureter and the bladder sphincter through its spastic components, and makes possible an exodus of bacteria as a result of paralysis of the musculature of the intestine. We believe today that the large majority of all postoperative retentions of urine in the bladder are produced not by the operation on the organs adjacent to the bladder, but by the preoperative and postoperative use of morphine. Very many surgical clinics have, therefore, entirely given up the use of morphine and limit themselves to substitution products. The German chemical industry, however, is endeavoring to remove this spasm-producing radical from the morphine molecule.

When we attempt to draw the therapeutic conclusions from these considerations of the pathology of the infections of the kidney, the first principle established is, that a severe disease of the kidney may develop under unfavorable conditions from even the most harmless appearing postoperative bacilluria or mild pyuria. Among these unfavorable conditions a motor insufficiency of the efferent urinary passages following many very different types of operation assumes the first place. Inasmuch as this stasis can be determined and treated only by catheterization of the ureters, a careful urological examination in all such cases is the most important requisite of medical knowledge and the physician's duty. It is worthy of mention, that in some cases even the drainage of the bladder by a simple catheter accomplishes also drainage of the kidney. On the other hand, pyelonephritis is still to a great extent outside the pale of our therapeutic ability, as we know little of the predisposing factors of its development. In this case, the therapy is limited to the intensive use of intravenous injections of urotropine. According to our view, a careful control of intestinal motility occupies the center of the stage in all prevention and therapy of colon bacillus infections of the urinary tract.

POSTOPERATIVE EVENTRATION*

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COMPLETE wound breakdown with an escape of part of the abdominal contents is a postoperative complication that happens without apparent rhyme or reason. No operator who does any amount of abdominal surgery will go for long periods of time without having it happen to a patient.

In a search through the literature very little has been found written on the subject. There is no unanimity of opinion as to the causative factors. It has been claimed that sepsis, hemorrhage into the peritoneum, faulty ligatures, poor approximation of the wound edges, excessive postoperative coughing with suture breaking, and syphilis lead to this condition.

Except for a persistent slight elevation of temperature, never more than two degrees one has no warning eventration will occur. When the wound sutures or clips are removed, the skin seems as far as inspection goes, healed by primary union. There is no bulging. The wound looks healthy. Dressings are applied and, as a rule, all thought of the wound is dismissed from the operator's mind.

About two or three days later the operator's house-man or nurse telephones him that the patient's wound has opened and intestines and omentum are spread over the abdomen. The patient may be in mild shock. Often the patient has suffered no sensation of moment.

When observed the wound edges seem dry and necrotic. There is no evidence of hemorrhage. Pus is absent.

As soon as the diagnosis of eventration has been made the attendant, interne or nurse covers the eviscerated contents with sterile gauze wet with warm boric acid solution. The patient is given grains ¼ of morphine sulphate. The operating-room is made ready.

The technic we offer for correcting this complication is not original.

The wound edges are freshened by rubbing them with sterile gauze sponges. The abdominal contents are returned within the abdominal cavity. Two strips of 2 to 4 in. iodoform gauze, each strip from 4 to 6 in. long, are sutured end to end with one interrupted plain catgut suture. This sutured end to end gauze is placed over the intestinal contents presenting through the wound, the ends protruding beyond the upper and lower wound angles. The wound is closed over this gauze using interrupted silkworm sutures. Each suture takes in every layer of the abdominal wall. It is poor practice to attempt closure in layers.

About the tenth day after resuturing, the gauze strip is removed. The plain catgut suture has been absorbed and by pulling on the end protruding from each angle of the wound the gauze comes away in two pieces. About five days later the silkworm sutures are removed.

After the resuturing, the patient is left flat in bed. A Harris or Murphy drip is instituted and continued for three or four days. Small doses of morphine are given for the first forty-eight hours.

As a rule the convalescence is uneventful. We have seen or heard of no second eventration following the closure we have outlined.

The writer has had five postoperative eventrations. All 5 cases were treated by this technic. All wounds subsequently healed. There were no fatalities.

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DANGERS IN THE USE OF HYPERTONIC SOLUTIONS IN THE TREATMENT OF BRAIN INJURIES*

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SINCE Weed and McKibbin[1] reported that the pressure of the cerebrospinal fluid could be reduced by the changes in brain volume, intracranial pressure, blood pressure and respiration following the intravenous injection of

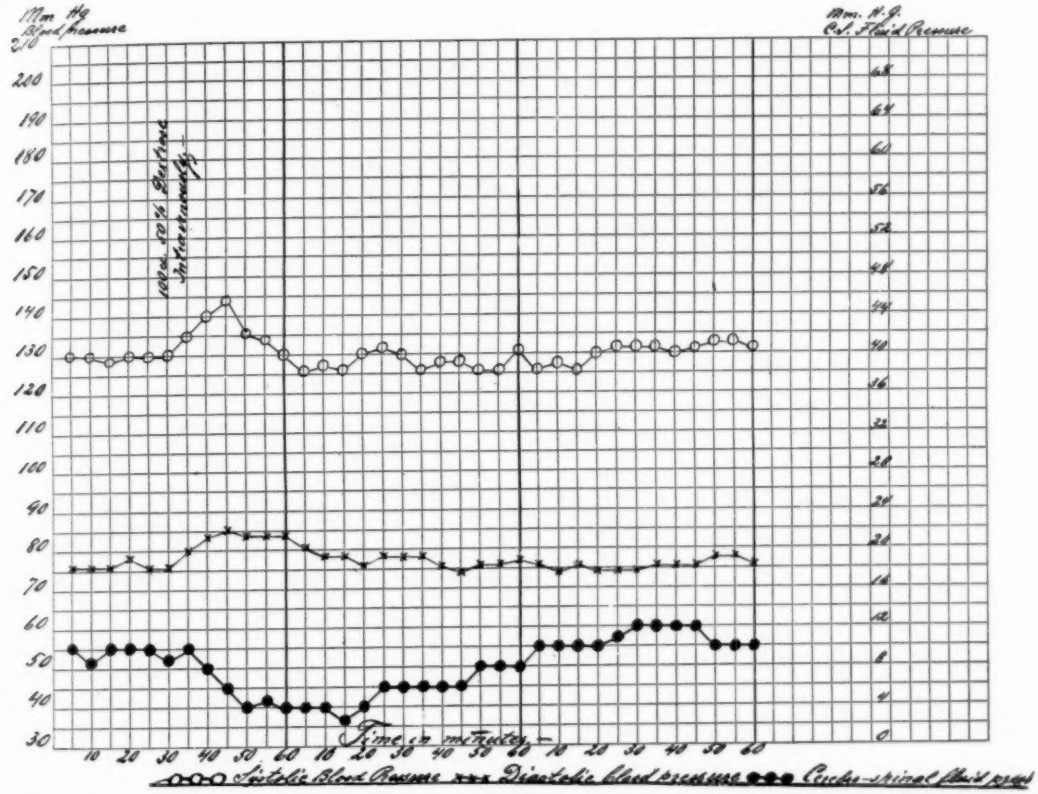


FIG. 1. Average curves, blood pressure and cerebrospinal fluid pressure of 4 normal individuals following intravenous injection of 100 c.c. of 50 per cent glucose.

the intravenous injection of hypertonic solutions, many clinical applications of this phenomena have been devised. Foley and Putnam[2] reported their experimental observations following the intravenous, oral, rectal and intraduodenal administration of 25 per cent sodium chloride solution. In each instance a distinct fall of cerebrospinal fluid pressure was noted. Comparing the results obtained from the intravenous method and the gastrointestinal route of administration, they found that the extent of the fall in the 2 cases was approximately equal. Sachs and Malone[3] investigated 25 per cent saline solution, using dogs as the experimental animal. They concluded that the method was a valuable aid in reducing brain volume in cases of increased intracranial pressure.

Foley[4] in a later communication reported his observations in the human subject, measuring the changes in the cranial pressure by the tension of a bulging decompression opening. Both the intravenous and oral methods of administration were used, the fall in the cerebrospinal fluid pressure being approximately the same in the 2 instances. He also calls

* Submitted for publication, April 2, 1930.

attention to the fact that there may be medullary depressant effects during or immediately following the intravenous

25 per cent saline solution the present study was undertaken.

Different concentrations of both glucose

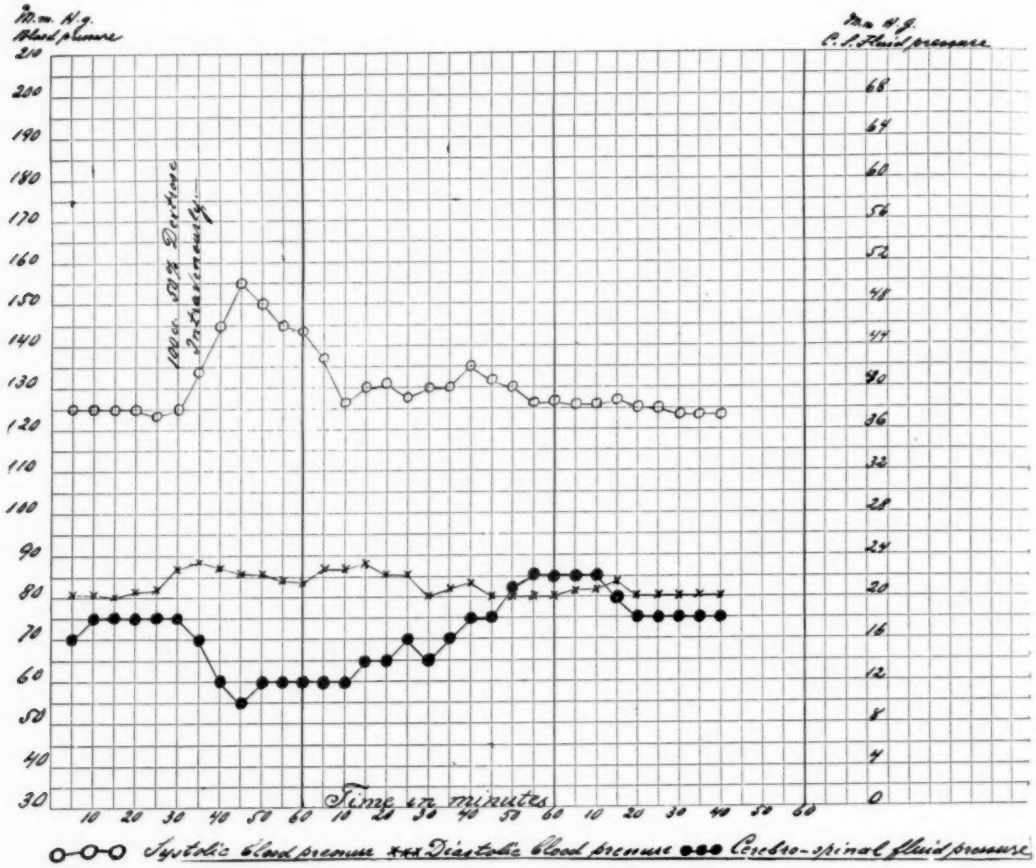


FIG. 2. Average curves, blood pressure and cerebrospinal fluid pressure of 5 patients, type 1 reaction.

injection of hypertonic solutions, which was not observed when the gastrointestinal route was used.

From the time of these publications, the methods of treating increased intracranial pressure due to brain hemorrhage and edema associated with trauma to the head began to change from the time-honored sub-temporal decompression or repeated spinal drainage to the intravenous and rectal injections of hypertonic solutions.

That great damage may be done by the routine use of so powerful a dehydrating agent as 25 per cent sodium chloride or 50 per cent glucose solution has been amply shown by the present clinical investigation.

Because of a fatal outcome following the intravenous injection of 60 c.c. of a

and saline solutions have been used, but it has been found by clinical experience that 50 per cent glucose in 75 c.c. to 100 c.c. doses produces the desired effect and causes fewer untoward reactions. Because of this fact glucose in such concentration was used in these experiments.

Subjects: All patients were adult males. Four with traumatic lesions of the extremities were used as controls. In each, the reduction of the cerebrospinal fluid pressure was about the same. The average of the 4 (Fig. 1) has been charted.

Thirteen patients admitted to the hospital because of injury to the head were studied. Four of these observations were not completed because of restlessness of the patients. Of the remaining 9, 8 had been in the hospital for periods ranging

from six to ten hours. These were unconscious or stuporous, but quiet and would remain in any position placed. The ninth was in deep coma, bleeding from nose and right ear, absent reflexes and fluid obtained from lumbar subarachnoid space had the appearance of venous blood.

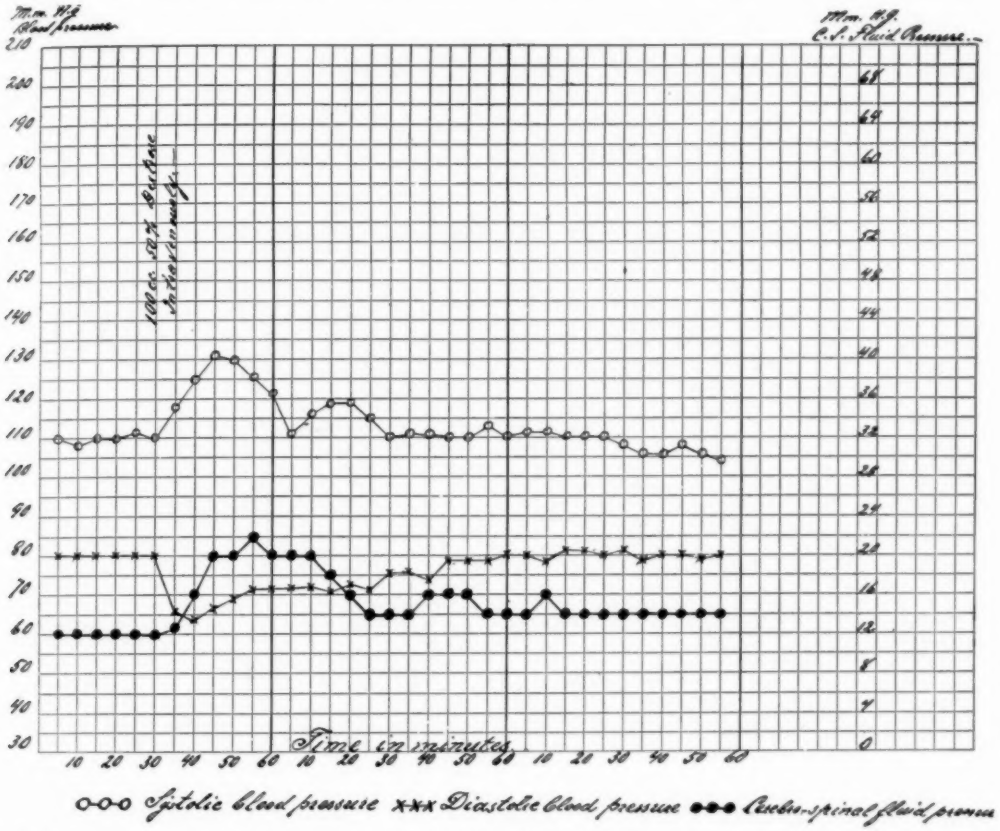


Fig. 3. Average curves, blood pressure and cerebrospinal fluid pressure of 3 patients, type II reaction.

Technic: Throughout the period of observation the patient was kept on the right side with lower extremities flexed, spine flexed and head placed on a pillow in normal relation to the trunk. A large bore spinal-puncture needle was introduced into the lumbar subarachnoid space and attached to a mercury manometer without loss of cerebrospinal fluid. The same size and length connecting tubing was used in each case. A sphygmomanometer cuff was attached to the left arm and diaphragm of a stethoscope fixed in left anticubital fossa. Blood pressure, pulse and cerebrospinal fluid pressure readings were taken every five minutes for twenty-five to thirty minutes before the intravenous injection of the 50 per cent glucose solution was begun. Readings were taken every five minutes for a variable period following the injection, the average being two hours and fifty minutes.

Results: In this small group of patients there were 3 distinct types of reactions.

In type I group, there were 5 patients who had a moderate decrease in their cerebrospinal fluid pressure associated with slight rise of blood pressure and pulse rate immediately after injection of the hypertonic solution (Fig. 2). There was also improvement of their mental state as evidenced by attempted motor activity and general appearance. None of these regained consciousness, although the cerebrospinal fluid pressure as measured at its lowest point was below the accepted normal. In all the cerebrospinal fluid was pinkish tinted with red blood cells.

In type II group, there were 3 patients

whose cerebrospinal fluid pressure showed a distinct rise following the injection of the hypertonic solution. This was associated with a rise of blood pressure and pulse rate (Fig. 3). The only change noted in their general condition were slight cyanosis of the skin and mucous membrane and increase in the thoracic respiratory excursion. This disappeared after a period of ten to fifteen minutes. These 3 subjects were thought to have severe brain injury. In each the cerebrospinal fluid contained blood in sufficient quantities to produce a cherry red color.

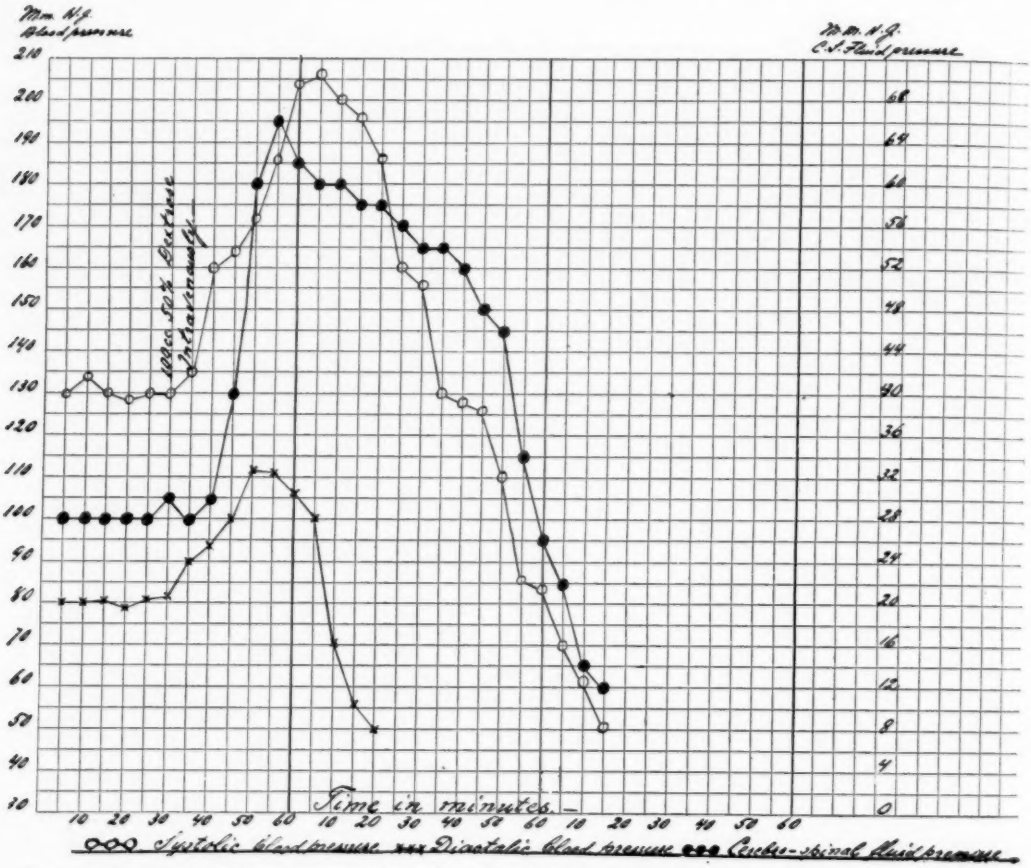


Fig. 4. Curves, blood pressure and cerebrospinal fluid pressure of 1 patient, type III reaction.

In type III group, there was 1 patient whose condition has already been briefly described. Almost immediately following the administration of the hypertonic solution there was a marked rise of blood pressure, cerebrospinal fluid pressure, and pulse rate (Fig. 4). The respirations were also accelerated, labored and irregular. After thirty-five minutes as indicated clearly by the chart, the blood pressure began to fall. This was associated with an extremely rapid pulse, rapid labored respirations and cyanosis. Death occurred one hour and fifty minutes following the injection of the hypertonic solution. At autopsy there was found lacerations of the under surfaces of the frontal lobes with large amount of subarachnoid hemorrhage (Fig. 5).

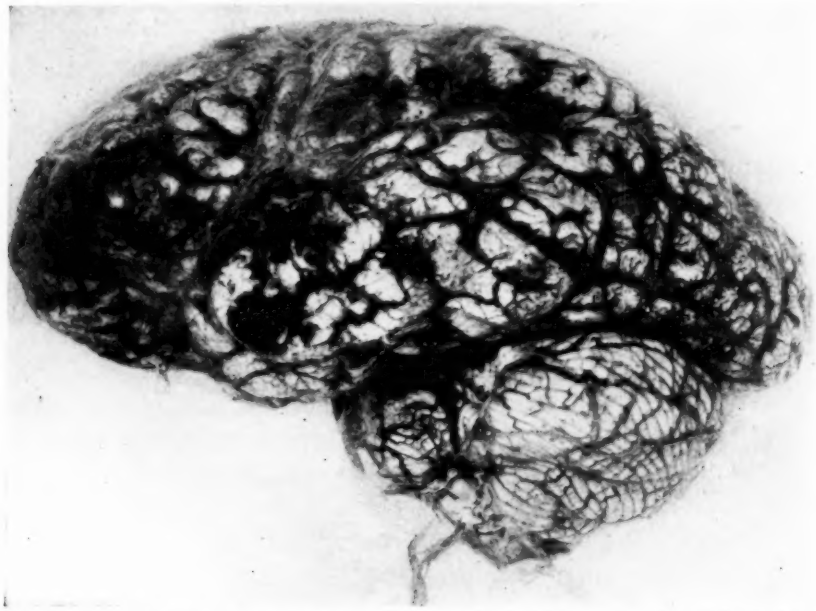
DISCUSSION

It would be impossible to draw any conclusions from such a group of cases, but for the fact that the author feels that they are representative of a group of head injuries in a large hospital where about 400 such patients are admitted annually. Many of these are in deep coma when admitted and die within a very short time regardless of what is done for them. Others seriously injured react after one to three hours, but die from twenty-four to

forty-eight hours regardless of what type of therapy is instituted.

These 2 loosely classified groups make up 18 per cent of the total number in 946 consecutive admissions. That increased intracranial pressure alone is not the only factor causing death can be demonstrated in most instances by repeated manometric readings. Often one finds the spinal or cisternal subarachnoid pressure only slightly above the accepted normal when at autopsy extensive cerebral laceration or contusion is present. From clinical and pathological observations it would seem that the terminal factor is intercellular and intracellular edema, particularly of the medulla.

With this pathological picture in mind the logical attempt at therapy would be to change the osmotic pressure of the blood by injecting hypertonic solutions thereby causing a decrease in brain tissue edema. A transitory improvement following such a procedure has been observed in many instances whereas harmful and even fatal results have occurred in others. It must be kept in mind that reducing increased cerebrospinal fluid pressure due to intracranial lesions without a recent break in the cerebral vascular system is quite a different problem from that due to hemorrhage and edema following trauma. In the former such as tumor, edema surrounding a tumor or edema secondary to trauma without evidence of hemorrhage, hypertonic solutions may be given with a certain degree of safety. In the latter instance where there are findings pointing toward recent blood vessel injury with subarachnoid hemorrhage, one should be very cautious about changing the osmotic pressure of the blood.



[Fig. 5. Photograph of brain of patient showing type III reaction. Note extensive subarachnoid hemorrhage.

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ALKALOSIS*

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IN presenting this paper it is our intention to discuss the early diagnosis and treatment of alkalosis, to emphasize the importance of early diagnosis, and to review a portion of the voluminous literature that has appeared on the subject during the last few years.

The term alkalosis covers a definite clinical picture which may be found in cases that are undergoing medical treatment as well as in postoperative cases. The terms acute paralytic ileus, acute gastric dilatation, gastric tetany and acidosis have been used in the past to denote the syndrome found in this condition.

The condition may occur in cases of intestinal obstruction, the severity varying with the acuteness and location of the obstruction; in cases of peptic ulcer where the patient has been receiving alkaline treatment; and as a postoperative complication following general anesthesia, and to a lesser degree, local and spinal anesthesia.

The patient is mentally alert and apparently unaware of the seriousness of his condition. However, he looks acutely ill, with cheeks sunken and eyes bright. He is apt to talk on trivialities. His skin is cold with beads of perspiration standing out on his forehead. As the disease progresses he shows fibrillary twitching of the hands and feet. The extremities are cold and clammy and the lips and nail beds are often cyanotic. The patient passes into a deep coma preceding death. Vomiting is an early and constant symptom, the vomitus being at first gastric and duodenal contents, and later being fecal in character, light brown in color and of foul odor. Vomiting at first is accompanied by retching but later becomes passive, apparently spilling from the patient's mouth. Hiccough is a frequent symptom, often being one of the first to develop. In the early stages the patient may pass gas and feces but soon there is complete cessation of intestinal movement with tympanites. The temperature is normal or subnormal with an antemortem rise. The pulse is weak and rapid from the start and the blood pressure is persistently low. The respirations are shallow and rapid. In short, the clinical picture is that of shock plus intestinal obstruction.

The red blood count is normal, becoming increased later due to dehydration from persistent vomiting. There is a leukopenia. The urine is usually scanty and on examination shows a trace of albumin and a few casts. The non-protein nitrogen in the twenty-four-hour specimen is markedly increased, often as much as 4 or 5 times the normal amount. The urinary chlorides are greatly reduced. Kidney function, as shown by dye excretion, is normal or slightly decreased.

The blood chemistry is characteristic and the most important of the diagnostic points. There is a constant increase in the non-protein nitrogen, a marked decrease in the chlorides and an increase in the CO_2 combining power of the blood.

The pathological findings are not diagnostic. The chief feature is a marked splanchnic engorgement. In the absence of obstruction the intestine is distended with fluid and gas. If an obstruction is present the intestine, if not ruptured, is much distended above the site of obstruction. The intestinal wall is dusky red and may present areas of gangrene. The lumen is full of foul smelling reddish brown fluid teeming with bacteria. The mucosa is deeply injected and presents ulcerations. Histologically the intestinal wall presents marked capillary engorgement and areas of necrosis. Some have described fatty changes in the liver and evidence of toxic nephritis.

The two outstanding features are:

* Read before the Brooklyn Surgical Society, December 6, 1929.

1. The intense toxemia.

2. The marked dehydration accompanied by a constant blood chemistry, consisting of a rise in non-protein nitrogen, a fall in the blood chlorides, and an increase in the CO_2 combining power.

For years numerous investigators have been studying this toxemia and although there is not complete agreement on the subject, the consensus of opinion appears to be that the causative toxin is found in the intestinal content above the obstructive point. The higher the point of obstruction, that is, the nearer it approaches the ampulla of Vater, the more severe is the clinical picture, and the more rapidly the case progresses to fatal termination. It has been shown that the contents from the intestines in obstruction, or from an obstructed intestinal loop, if injected into a laboratory animal, will produce the identical picture of toxemia with death in a few hours. Furthermore, if a dog with a duodenal fistula is given an injection of either the intestinal contents from an obstructed loop, or the intestinal contents from a dog which has been injected with the contents of an obstructed loop, this toxin will be recovered from the duodenal fistula within two hours after the injection, and will be equally as toxic to another animal as were either the contents from the obstructed loop or from the second dog injected. This process can be repeated indefinitely. Cooper,[1] after a most exhaustive study of the literature pointed out the following facts concerning the properties of this toxin:

1. It is held back by an earthen filter but not by paper.

2. It does not pass a semipermeable membrane.

3. It is not destroyed by boiling.

4. It is not destroyed by long digestion with pancreatic juices (seven days).

5. It is not destroyed by autolysis with intestinal mucosa (for months).

6. It is not destroyed by erepsin.

7. It is probably destroyed by hydrolysis with 5 per cent sulphuric acid for six hours at 100°C.

8. Repeated precipitation removes its toxicity.

9. It loses its toxicity if dialyzed against physiologic sodium chloride for several hours.

10. It loses its toxicity by repeated filtrations.

11. It loses its toxicity by long standing at freezing temperatures.

12. It is soluble in water.

13. It is precipitated by 5 volumes of alcohol.

14. It is precipitated by albumin.

15. It is precipitated by 60 per cent ammonium sulphate solution.

16. It does not stimulate pancreatic digestion.

Although the toxin has been demonstrated in the contents of the intestine, the origin of the toxin is in doubt. In obstructed loops the toxin has been demonstrated to be both in the blood and lymph draining from the loop. However, the blood of an animal with marked toxemia from obstruction is non-toxic to another animal even though the entire blood is transfused.

In obstruction in the newborn or in sterile closed loops in experimental animals, this toxin is apparently absent, which fact would seem to point to a bacterial origin. Against this, however, is the fact that the higher the obstruction the more severe the toxemia, while the bacterial count is comparatively low in the upper intestines as compared to that in the colon. If the toxemia is bacterial in origin, obstruction of the large intestine should therefore be much more severe than that of the duodenum, which we all know is not the case. Moreover, death from a toxemia apparently identical with that in obstruction is seen in acute strangulation, in complete extirpation of the suprarenals, in bilateral ligation of the ureters, thrombosis of the portal vein and distemper.

What is the origin of this toxin? Experimental work has rather conclusively ruled out the stomach as a source. Elimination

of the bile does not alter the picture. The pancreas cannot be a factor as the same picture is presented in depancreatized dogs. There is much difference of opinion as to whether the secretion of the intestinal mucosa may be the causative factor. There are also many varied opinions as to the part bacteria play, and those who favor it must explain:

1. Why is high obstruction more rapidly fatal than low obstruction?

2. Why does toxin appear with such rapidity (within three hours) after obstruction?

3. Why does toxin appear almost immediately in the intestines of dogs that have received injections of it?

4. Why does the toxin appear in acute pancreatitis, thrombosis of the portal vein, bilateral suprarenalectomy, bilateral ligation of the ureters, and distemper?

Some think that the toxin may arise from partly digested food and that the toxin is derived from protein digestion. The majority of observers think that the toxin is absorbed through the injured gut mucosa.

We shall consider the second outstanding feature of this condition namely, dehydration and blood picture.

Neither the dehydration nor the altered blood chemistry can be considered as cause of death, since death occurs in strangulation and in the other conditions enumerated above without dehydration or a change in the blood chemistry. The dehydration is a result of the persistent vomiting and is directly proportional to it. Also the decrease in the blood chlorides is directly proportional to the vomiting. The loss of the Cl or acid radical causes a disturbance in the acid-base balance with a resulting alkalosis. As a result of the loss of Cl the excess sodium radical is taken up by the acid phosphates of the tissues instead of being neutralized by the Cl from the intestinal tract to form NaCl.

Hence the decrease of blood chlorides is directly proportional to the loss of Cl by vomiting. As a a result of the loss of the acid radical Cl, there is a rise in the carbonic acid radical of the blood, this being a natural attempt of the body to maintain acidity of the blood at its proper level. The increase in the CO_2 combining power of the blood varies inversely as the decrease in the blood chlorides and is also directly indicative of the degree of alkalosis.

The rise in non-protein nitrogen is the result of tissue metabolism with destruction of the proteids. It cannot be explained by dehydration since it has been repeatedly shown that the increase in the urea is relatively many times greater than the degree of dehydration. Further, it cannot be explained by retention, since the kidney function is normal and the urinary output is normal or only slightly decreased. The cause of this proteid destruction is unknown. However, the increase in the non-protein nitrogen in the blood is directly proportional to the decrease in the blood chlorides and to the increase in the CO_2 combining power, and all three respond correspondingly to the same line of treatment.

So much for the toxemia and dehydration, but what has happened to the unobstructed bowel that fails to transport fluids and gas? Alvarez and Hosoi[2] showed by experimental work on animals that section of the spinal cord distal to the fourth dorsal segment caused such a strong stimulus to peristalsis that after a few hours the bowel tends to empty itself with repeated rush waves. In animals with the terminal ileum injured they saw few rush waves and in some they seemed to be stopped entirely, even though the lumen of the bowel was unchanged and the rhythmic movements were active and the muscles efficient. Alvarez and Hosoi believe therefore, that we must find a new term for the type of ileus which is not due to mechanical obstruction or narrowing of the gut lumen. It is not paralytic because the muscle is active and apparently in good working order,

and it is not dynamic since there are none of the white ring-like contractions that sometimes obstruct the bowels in children. They offer the explanation that there is a flattening, or reversal, of the normal gradient of forces, and an excess of inhibition, and suggest the term flat gradient ileus for this type of condition.

TREATMENT

In comparison with the many and varied opinions as to the cause and origin of the toxemia in alkalosis, we find that in the treatment of the condition most observers are agreed, at least upon the essentials.

Care should be used in the administration of alkaline drugs in the treatment of peptic ulcer especially in the presence of pyloric obstruction or kidney damage.

In preoperative cases where there is intestinal stasis and no emergency exists, medical treatment consisting of repeated stomach lavages and the administration of NaCl should be carried out until the blood chemistry has been brought within normal limits.

Preoperative purgatives are to be condemned as they weaken the muscles and disturb the mechanisms that remove the gas.

Alvarez and Hosoi[2] demonstrated that distending and irritating enemas especially with glucose solution tended to reverse the gradients and produce more back pressure and reverse peristalsis.

As little morphine should be used as possible as in many persons it increases the tendency to reverse peristalsis. Barbital derivatives do not upset the gradients and are probably the best substitutes we have for morphine.

Some think that ether upsets the gradients and should be replaced by other forms of anesthesia. However, we have seen several cases of alkalosis develop following both spinal and local (procaine) anesthesia.

Unnecessary and rough handling of the abdominal viscera is advised against.

As to the various drugs that are commonly in use, atropine and strychnine are unreliable and ineffective. Nicotine would be an ideal drug if it were not so highly toxic, as it blocks the passage of nervous impulses through the sympathetic ganglia. Pituitrin, when given in large doses of physiologic NaCl will stimulate peristalsis but it is as apt to do harm as good. Eserin is not reliable. Alvarez and Hosoi[2] state that choline is a drug that gives great promise. Von Magnus[3] and his pupils in their experimental work have much evidence to prove that it is a normal constituent of intestinal muscles and is concerned in some way with maintaining peristalsis. The drug must be chemically pure as it tends to break down into toxic substances. Merck and Kahlbaum supply it in ampules containing 600 mg., and put up in this manner it will keep for several years. For a man weighing 60 kg., the 600 mg. should be added to at least 250 c.c. of physiologic salt solution and run into a vein so slowly that the process will take at least seventeen minutes. If the patient complains of flushing, dyspnea, dizziness and salivation, and the pulse becomes slow, the container should be lowered. We have had no experience with this drug.

As to food some think that these patients should be fed for the purpose of stimulating peristalsis, and suggest that meat be given them as it probably has the most stimulating effect on the tone of the gastrointestinal tract. Our experience with feeding has led us to believe that it only aggravates the condition unless the toxemia and vomiting have previously been relieved by lavage and NaCl.

Much difference of opinion exists as to the value of the antitoxin of Bacillus welchii. Morton and Stabin,[4] in their experimental work found that the toxin of B. welchii has a bearing on intestinal obstruction and that the antitoxin appears to be specific in its action, since other forms of antitoxin are not effective. Owings and McIntosh[5] found that the life of dogs in high obstruction was not

prolonged by antitoxin of B. welchii and immunization of dogs of the toxin of B. welchii did not prevent a fatal outcome in those given a minimum lethal dose of loop toxin. Stabins and Kennedy[6] experimentally recovered the organisms of B. welchii from the jejunum 10 in. distal to the ligament of Treitz in 17 per cent of their series at the time of artificial obstruction at this level. After the development of toxic symptoms the organism was recovered proximal to the obstruction in 94 per cent of their animals. Williams[7] reports very favorably upon the use of the antitoxin at the St. Thomas's Hospital. It was administrated to 18 of the most severely ill of a series of 256 consecutive cases of obstruction following acute peritonitis. There were three deaths, making a mortality rate of 1.17 per cent. A control series of 111 cases at the same hospital which were not given the B. welchii antitoxin showed a mortality rate of 6.3 per cent. The antitoxin was given in addition to operative relief in 54 cases of acute intestinal obstruction, and there was a mortality rate of 9.3 per cent as opposed to the rate of 24.8 per cent in a series which did not receive antitoxin. Bower and Clark[8] report favorably upon its use, having administered it in 9 cases of acute intestinal obstruction, 11 of diffuse suppurative peritonitis, and 5 of suppurative cholecystitis. They found their patients less restless; the pulse rate was diminished, the temperature reduced, abdominal distention became less, and bowel movements began earlier.

Bonney[9] in 1910, was the first to advocate drainage of the jejunum, and since that time jejunostomy has been used with rather indifferent success. Van Beuren and Smith, [10] in reviewing the cases of enterostomy performed in acute ileus at the Presbyterian Hospital from 1914 to 1923, and the Roosevelt Hospital from 1910 to 1919, showed a higher mortality rate in the enterostomized cases than in the entire group, but were of the opinion that enterostomy had been reserved as a last resort in the most critical cases, and therefore had not a true bearing on the efficacy of the treatment. Dudley[11] states that obstruction of the paralytic type accounts for 40 per cent of the deaths following operation for acute appendicitis in the presence of diffuse peritonitis, and believes that jejunostomy is the most efficient means of combating the toxin. Orr and Haden[12] found enterostomy of no avail in fulminating general peritonitis. In peritonitis of the lower part of the abdomen it is life-saving and in all doubtful cases should be done. Bartlett[13] states that the high mortality reported following enterostomy is the inevitable concomitant of its use in patients already dying. He uses spinal anesthesia in determining when to perform an enterostomy. If within fifteen minutes after the injection of the spinal anesthetic, passage of gas and feces and disappearance of abdominal distention does not occur, enterostomy should be performed immediately, thus taking advantage of the anesthesia already produced. Longer delay permits progressive depletion of the NaCl and water reserves, interference with circulation of the gut, absorption of toxins from the intestines, and advancement of those other processes which singly or in combination cause death.

Spinal anesthesia in the treatment of intestinal obstruction has many proponents. Greig[14] reported favorably on its use in 400 cases. Studdiford[15] especially recommends it in dynamic ileus but also uses it in all types of obstruction to be followed by operation if necessary. Markowitz and Campbell[16] have used spinal anesthesia in the laboratory on dogs after producing ileus by chemical or traumatic means, and have observed peristalsis carefully by means of barium meals and the fluoroscope, when they have noted that peristaltic movements commenced soon after the spinal anesthesia and persisted in some instances for over twenty hours. They offer the conception that paralytic ileus is a reflex inhibition of bowel movements. Starling[17] states that stimulation

of the splanchnic nerves causes complete relaxation of the intestine, while stimulation of the vagus causes increased contraction following a brief period of relaxation. He also suggests that the relaxed condition of the intestine in many abdominal conditions is probably due to reflex stimulation of the splanchnic nerves which nullifies the motor action of the vagus.

The probable explanation of the effect of spinal anesthesia in ileus is that splanchnic inhibitory reflexes are blocked so that the vagus motor reflexes have full power. It is well to keep in mind that spinal anesthesia will not be effective if the patient is under the influence of morphine.

The efficacy of the treatment of alkalosis by the use of hypertonic solution of NaCl has been proved both experimentally and by clinical use. NaCl in 2 to 25 per cent solution intravenously acts as a strong intestinal stimulant. It has been shown beyond a doubt that water alone will not influence alkalosis, and also that KCL, NH_4Cl, $CaCl_2$, and $MgCl_2$ are not only useless but increase the severity of the alkalosis. Anderson and Rockwood[18] reported 90 cases of low blood chlorides associated in most cases with abdominal lesions treated with hypertonic saline solution intravenously with a reduction of the operative mortality of more than one-half. Foster and Hausler[19] brought out the fact that loss of chlorides is apt to occur in what they call the "critical period," which corresponds with the healing period of intestinal wounds in experimental animals, and lasted four or five days after operation. They therefore suggested keeping the chlorides at their proper level for five days. This conception we think is well founded and in practice has given us excellent results. It has been our custom to give 1000 c.c. of normal saline intravenously plus 100 to 500 c.c. of 2 per cent NaCl solution and 40 to 50 gm. of glucose daily. The glucose is given in 5 per cent solution with the normal saline. This we have found controls the toxemia in the average case quite effectively.

The treatment should be checked by blood chemistry examinations whenever possible, but where such procedures are not easily available the course of the treatment may be checked by the clinical picture which improves as the chlorides approach normal. In experimentally produced general peritonitis Orr and Hayden[12] were able to keep animals alive 3 times as long with ileostomy plus 1 per cent NaCl as with ileostomy alone, and they state that no treatment for peritonitis should be given to the exclusion of large quantities of saline solution to relieve the dehydration and hypochloremia.

In addition to the intravenous therapy it is advisable to drain the upper intestinal tract of its toxic content, either by repeated gastric lavages or duodenal drainage. It is surprising how quickly the hiccough, the vomiting, and the clinical picture of shock subside after a few hours of duodenal drainage. However, we must keep in mind that in draining the intestinal tract we are predisposing to further dehydration and hypochloremia and must make up for it by giving saline intravenously.

If tetany develops, calcium chloride should be given in addition to the NaCl. This is administered most effectively in 5 c.c. doses of 10 per cent solution intravenously, and repeated every two hours until controlled.

Formerly the accepted treatment in cases of persistent vomiting in intestinal obstruction, postoperative ileus and allied conditions was the use of sodium bicarbonate, either intravenously or by proctoclysis, and the term acidosis was generally accepted for the clinical picture. With our present knowledge of the blood chemistry findings further attempts at alkalinization are certainly contraindicated. Whatever benefits may have been observed following the use of sodium bicarbonate was probably due to the introduction of the sodium radical.

It is quite natural for a patient who has had an abdominal operation to prefer to lie quietly in bed on his back, and those

with tympanites usually insist on having the chest and head elevated. Unless there are some contraindications we believe that patients should not only be encouraged, but should be forceably moved from side to side, which will tend to shift the gas from one part of the intestines to another. Moreover, it is reasonable to expect that gas trapped at the splenic flexure might be thrown into the rectum where it could be passed, by simply elevating the hips above the mid-portion of the body.

The following cases are reported:

Case I (33442). White, obese, male, age thirty-four years.

Admitted to hospital on December 6, 1928, with a history of having developed generalized abdominal pain four days previously which soon settled in his right lower quadrant. Temperature was 101.6, pulse 120 and respiration 18. The leukocyte count was 13,400 with 91 per cent polymorphonuclears. Urine was positive for albumin and hyaline casts. A gangrenous appendix was removed under ether anesthesia on the day of admission. On the second postoperative day he complained of belching and abdominal distention and during the night began to vomit. The stomach was lavaged and the Rehfuss tube left in place. Glucose was given intravenously and normal saline by hypodermoclysis. Strychnine sulphate and pituitrin were given repeatedly without any appreciable results. On the fourth postoperative day a bronchopneumonic patch was found at the right base. Glucose intravenously, normal saline by hypodermoclysis and proctoclysis were continued daily, and the first bowel movement was secured on the seventh postoperative day. Convalescence from there on was uneventful except for a colon bacillus infection of the abdominal wall. Had hypertonic saline been given intravenously instead of normal saline by hypodermoclysis recovery undoubtedly would have been much more rapid. Blood chemistry examinations were not done in this case.

Case II (32892). White, male, fifty-five years of age, was admitted on October 30, 1928, with a diagnosis of recurrent left inguinal hernia and old fracture deformity of left clavicle. Urine was reported as albumin positive 1 plus and hyaline casts 1 plus. Repair of left inguinal hernia was done on December 19, 1928. Thirty-six hours after operation complained of distention, was nauseated and vomited a small amount of greenish material. Gained some relief from an enema and rectal tube. Was given 1000 c.c. of normal saline intravenously. Vomited large amounts of greenish fluid on the following day and duodenal drainage was instituted and 1500 c.c. of normal saline given intravenously. On December 23, four days after operation, blood chemistry showed non-protein nitrogen 28 (mg. per 100 c.c.) and chlorides 363, (mg. per 100 c.c.) and he was given 1000 c.c. of normal saline intravenously. On the following day vomiting ceased and bowels moved well. On December 25 he again complained of marked abdominal distention and was given 1500 c.c. of normal saline and on the following day the blood chemistry showed non-protein nitrogen 18.9 (mg. per 100 c.c.) chloride 400 (mg. per 100 c.c.) and CO_2 combining power 53 (volumes per cent). On this date he was given 100 c.c. of 2 per cent NaCl and 500 c.c. of normal saline after which distention disappeared, flatus was passed and patient went on to an uneventful recovery. Strychnine, atropine, physostigmine, pituitrin and numerous enemas were given during the several days without any appreciable improvement. This case is cited to show that sufficient NaCl was not given early to control the toxemia.

Case III (4415). White, male, forty-three years of age, entered the hospital for treatment of a right inguinal hernia of one year's duration. Physical examination was negative except for a moderate sized right inguinal hernia and a markedly relaxed ring with strong impulse on the left side. The urine showed 1 plus hyaline casts but otherwise was negative. The patient had had some trouble with his appendix previously and requested that it be removed at the same time that his hernias were repaired. Both hernias were repaired under procaine anesthesia on October 2, 1929 and the appendix removed through the right hernia incision, the three operations requiring one hour and twenty-five minutes. On the morning following operation the patient complained of considerable distention. He had been given three hypodermics of morphine sulphate gr. ¼ for pain in the preceding twenty-four hours. Early in the second postoperative day he suddenly went into a state of shock and vomited a large

amount of dark brown fluid. He was placed between blankets and artificial heat was applied. The stomach was lavaged and the patient given saline and glucose intravenously, after which he reacted well. On the third postoperative day the patient was still considerably distended and had hiccough. Blood chlorides were 445 (mg. per 100 c.c.); saline and glucose were given intravenously. Condition was worse on the fourth postoperative day and the blood chemistry showed nonprotein nitrogen 28 (mg. per 100 c.c.) chlorides 429 (mg. per 100 c.c.) and CO_2 combining power 44 (vol. per cent). A jejunostomy was performed under procaine anesthesia with the release of a large amount of gas and foul smelling fluid. On the fifth postoperative day he was not improved and the jejunostomy tube was draining very little. Saline and glucose were repeated intravenously. On the sixth postoperative day there was marked dyspnea; temperature, pulse and respiration were markedly elevated. He passed a little gas per rectum and about 1500 c.c. of fluid fecal matter through the jejunal tube. The blood chemistry at this time showed nonprotein in nitrogen 28 (mg. per 100 c.c.) chlorides 445 (mg. per 100 c.c.) and CO_2 combining power 48 (vol. per cent). Glucose and saline were given intravenously and a cecostomy performed under procaine anesthesia as a last resort. The patient went into coma and died a few hours later. The autopsy findings were: Postoperative atelectasis, both lungs; chronic adhesive pleurisy; hypostatic bronchopneumonia; pulmonary edema; myocardial degeneration; cardiac dilatation; postoperative paralytic ileus, degeneration of the parenchymatous organs; adenoma of the prostrate; and passive congestion of the viscera.

Case iv (31404). White, male, age thirty years. Has a history of "stomach trouble" for past six years; appendix was removed in 1923. He has had several courses of Sippy diet. Had an attack of hematemesis and tarry stools one year ago, since which time he has been very weak. While on Sippy diet under observation in the hospital on July 21, 1928, he developed tetany which was controlled by normal saline and glucose intravenously, and calcium lactate grains x by mouth every hour. Gastric resection for obstructing pyloric ulcer was performed on August 8, 1928, from which the patient with the aid of glucose and saline intravenously made an uneventful recovery.

Case v (33744). White, male, age thirty-one years, admitted to hospital with a history of pain in right chest of one month's duration. *X*-ray showed a dense shadow in the axillary portion of the right lung field whose convex border, together with evidence of pleural thickening in the adjacent costophrenic sinus, was suggestive of an old encapsulated pleural exudate. The adjacent lung markings suggested atelectasis. Patient had an occasional chill, and temperature was of the septic type, ranging from 100 to 104°F. Thoracotomy was performed on January 8, 1929, which revealed a trabeculated cavity filled with blood clots. Drainage was blood tinged for several days, gradually becoming purulent. Temperature continued to be of the septic type and fourteen days after operation he developed hiccough and vomited. Hiccough continued more or less continuously for five days and the patient became delirious. At this time the blood chemistry showed non-protein nitrogen 28 (mg. per 100 c.c.) and chlorides 346 (mg. per 100 c.c.). He was given 250 c.c. of normal saline and 200 c.c. of 2 per cent saline intravenously which was repeated on the following day, after which his symptoms rapidly disappeared and convalescence was uneventful from then on.

Case vi (4121). White, male, age thirty-one years. Gives a history of typical gall bladder colic over a period of ten years. A chronically diseased gall bladder with a stone 1 cm. in diameter blocking the cystic duct, and the appendix were removed under spinal anesthesia on November 7, 1929. Twenty-four hours after operation patient vomited a large amount of brownish fluid. Blood chemistry showed non-protein nitrogen 15 (mg. per 100 c.c.) and chlorides 470 (mg. per 100 c.c.). Stomach was lavaged and hypertonic saline and glucose given intravenously with relief of all symptoms and an easy convalescence from then on.

Case vii (5334). White, male, thirty-six years of age. Appendectomy with exploration was performed under spinal anesthesia on November 26, 1929. Twenty-four hours after operation patient was comfortable except for some gaseous distention which was relieved by an enema. Forty-eight hours after operation he began to hiccough, became distended, and vomited large amounts of greenish fluid. Blood chemistry showed urea nitrogen 22.5 (mg. per

100 c.c.) chlorides 429 (mg. per 100 c.c.) and plasma CO_2 51 (volumes per cent). After gastric lavage and hypertonic saline and glucose intravenously patient passed flatus freely and the abdomen became soft.

In conclusion we wish to make a plea for the early recognition of the toxemia associated with paralytic ileus. Those who have to do with the post-operative care of patients should be on the lookout for the early signs that precede the characteristic changes in the blood chemistry. These are usually: a feeling of fullness in the epigastrium, belching, heartburn, hiccough, a craving for water, and a decrease in the urinary output. It is at this stage that treatment terminates so happily, both for the patient and the surgeon.

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CEMENT-STARCH MIXTURES AS A SUBSTITUTE FOR PLASTER OF PARIS IN THE TROPICS*

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DETERIORATION OF PLASTER IN THE TROPICS

SURGEONS working in tropical countries where the humidity is high often witness the rapid deterioration of unused plaster of Paris, resulting from absorption of moisture. This is particularly noticeable when the contents of 5 or 10 lb. containers are only partly consumed, as often happens when the use of plaster is not a daily or even a weekly occurrence.

In these circumstances, it is often found that the plaster has lost its smooth amorphous character and it becomes coarsely lumpy or mixed with hard, irregular granules. This deterioration is sometimes not apparent until the bandages are applied, when it is observed that the solid portions of the plaster remain dry and unaffected by immersion; the layers of bandage fail to cohere, and the resulting cast readily bends or cracks. Some grades of plaster appear to undergo this important change without exposure and as a result of aging alone, as the alteration may be seen in plaster taken from freshly opened containers which have been hermetically sealed. There is evidence, therefore, that such aging of hermetically sealed plaster takes place more rapidly in tropical localities than in climates where there is less moisture in the atmosphere.

SPECIAL METHODS ADOPTED IN USING PLASTER OF PARIS

To overcome these difficulties the following rules are commonly observed in the tropics: (1) Coarsely deteriorated plaster is discarded. (2) Doubtful plaster is used very liberally in rolling bandages, and about 50 per cent more is applied than in temperate climates. (3) Standard gauze bandage is used instead of crinolin or other wide-meshed material, in order to hold the increased plaster content. (4) Additional plaster is liberally applied during the application of a cast. (5) Reinforcements of metal or wood are frequently placed at points of stress, particularly in a hip cast. To assure drying, water at 110°–120°F. is employed in the bandage basin and a half teaspoonful of salt is added to it.

VARIETIES OF PLASTER OF PARIS

Certain French preparations of plaster marketed in 500-gm. sealed tins are entirely free of the objections mentioned. These products are of the finest quality and possess an added advantage in being packed in comparatively small containers. Best grade French plaster of Paris, however, is not always obtainable; and, moreover, in some countries a high import duty prohibits the economic use of this product.

SUBSTITUTE FOR PLASTER

A common indication of the necessity for providing a plaster substitute is the discovery that all of the plaster on hand has deteriorated to a degree which makes its effectiveness doubtful. This experience has probably been the lot of most surgeons working in isolated localities in the tropics where quick access to a supply of fresh plaster is not always available. When such an emergency situation is encountered, fixation by splinting or extension is not always a satisfactory alternative. The writer has found that mixtures of Portland cement with starch, with certain reservations, fulfill the requirements for an emergency or semi-permanent substitute for plaster of Paris.[1]

THE CEMENT-STARCH CAST

For most purposes, a mixture of equal parts by volume of Portland cement and

[1] Ryerson, E. W. The celluloid cast described by Ryerson is expensive and requires several days to complete. *J. Bone & Joint Surg.*, 8: 517–518, 1926.

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laundry starch will prove satisfactory. These materials are freshly mixed, triturated if lumpy, spread thickly on a gauze

Fig. 1. Cement-starch cast used for fixation and extension in fracture. Dislocation of third and fourth cervical vertebrae. When removed one month after application, this cast was in good condition. Composition: Cement 1 part; starch 2 parts.

bandage, rubbed smooth and loosely rolled. The bandages are placed in warm water (salt is omitted), gently compressed to insure exit of air bubbles and applied exactly as are plaster bandages. Cement-starch powder and water are thoroughly rubbed into the growing cast. The finished model is smoothed and polished with starch-and-water paste. Casts for the arm or leg should be at least 1 cm. thick after rubbing; and for the body, thigh and hip, they should be proportionately heavier.

For a light-weight cast, subjected to little strain, the mixture may be made of two parts by volume of starch to one part of Portland cement; while for a heavy cast, which may be subjected to considerable tension, the proportion may be reversed.

These casts generally require from twenty-four to thirty-six hours to become thoroughly dry and hard. New types of quick-hardening cement materially reduce the time of setting. During this period, immobility must be assured by cooperation of the patient. Drying may be hastened by the application of hotwater bottles to the cast, by exposure to sunlight, by hair-drying apparatus, or by utilizing one or more 200 cp. electric bulbs.

COMPARISON OF CEMENT-STARCH WITH PLASTER OF PARIS

Casts made in the manner suggested compare favorably with those constructed of the ordinary grades of domestic plaster of Paris or dental plaster. They are made of materials which do not deteriorate and which are always available. They are heavier and somewhat softer, and tend to become more moist than plaster casts. They bend readily, but are not so prone to crack. They appear to wear as well as ordinary plaster casts and are easier to remove in either the dry or wet state. Removal is best accomplished after soaking the cast in water to soften and dissolve its starch content. The moistened cement bandage is easily cut with a cast knife or cutter.

Economically, the cement-starch cast is of decided advantage. For example, in Guatemala, the cost of Portland cement is $0.0132 per pound, and of starch is $0.119 per pound; while plaster of Paris (French grade) is $0.30. Furthermore, there is a large percentage of loss from deterioration when ordinary plaster of Paris is used; this must be taken into consideration in making an estimate of the comparative gross cost of the product.

CONCLUSIONS

1. Ordinary grades of plaster of Paris are subject to rapid deterioration in tropical climates, which renders them unsuitable for exacting use.

2. High grade French plaster of Paris, packed in small containers, is stable and should be given preference when possible.

3. Mixtures of Portland cement and starch have been used satisfactorily as substitutes for plaster of Paris. The materials are cheap and readily available, and are not subject to deterioration.

4. The cement-starch cast compares quite favorably with casts made of the ordinary grades of plaster of Paris and is superior to those made of deteriorated plaster.

INTRAVENOUS UROGRAPHY

BETTER VISUALIZATION WITH COMPRESSION*

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HISTORICAL

SINCE 1897 when Tuffier first suggested the use of an opaque internal catheter, the profession has been floundering about in an effort to reach the ideal method of clearly demonstrating the outline of the genitourinary tract on the *x*-ray film. The interim between 1897 and 1912 is now of historical value only. Such men as Klose, Voelcker and von Lichtenberg, Keyes, Uhle and Pfahler, Doderlein and Kronig, Burkhardt and Polano, Dietlen, Kelly and Lewis, Baker, Oehlecker, Stanton and Bruce, Thomas, Braasch, Kidd, Fowler, Schramm, Childs and Spitzer, Alberran and Ertzbischoff, Nogier and Reynard, will always be remembered as the true pioneers of urography, through whose efforts the foundation of this great work was laid.

From 1912 to 1923, the common practice of injecting a 12 per cent sodium iodide solution through the catheter seemed the standard method of choice for this work. Urology had now become an independent field of medicine, and urologists seemed satisfied with this method. But in 1923, unrest again prevailed, and now knowing the true limitations of the retrograde method, a new technic of visualization by the intravenous route was sought. The names of Sutherland, Scholl, Osborne, Rowntree, Rosenstein, von Lichtenberg, Volkmann, Lenardouzzi, Pecco and Roseno will always be associated with the newer thought. Although unsuccessful in their

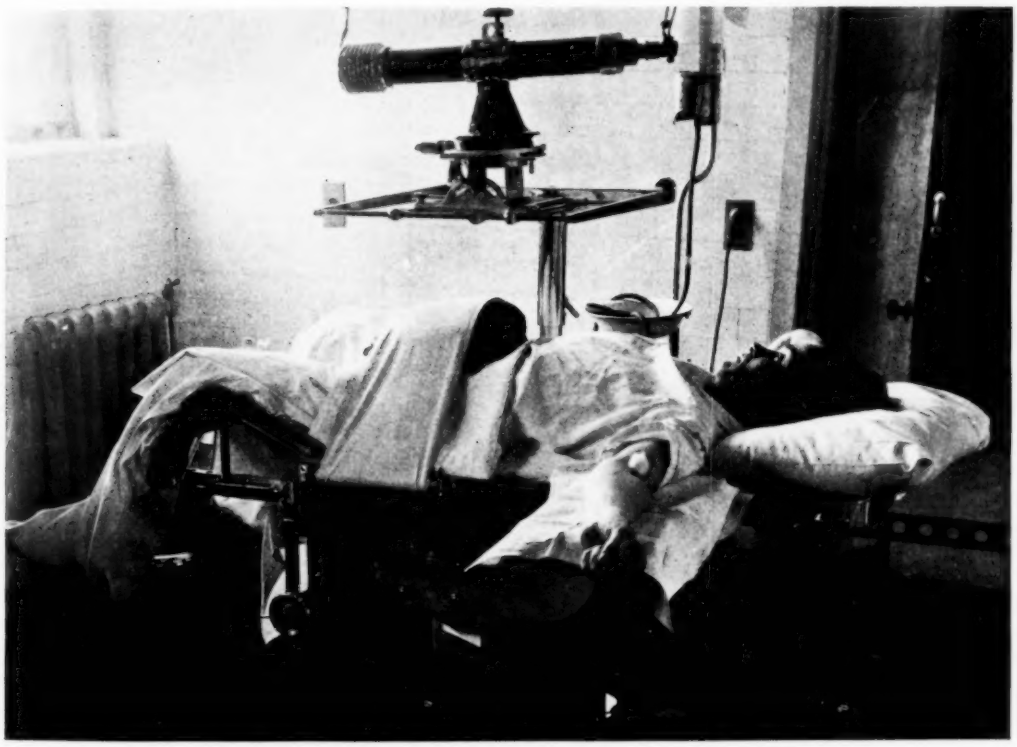

FIG. 1. Hickey bag employed in intravenous urography.

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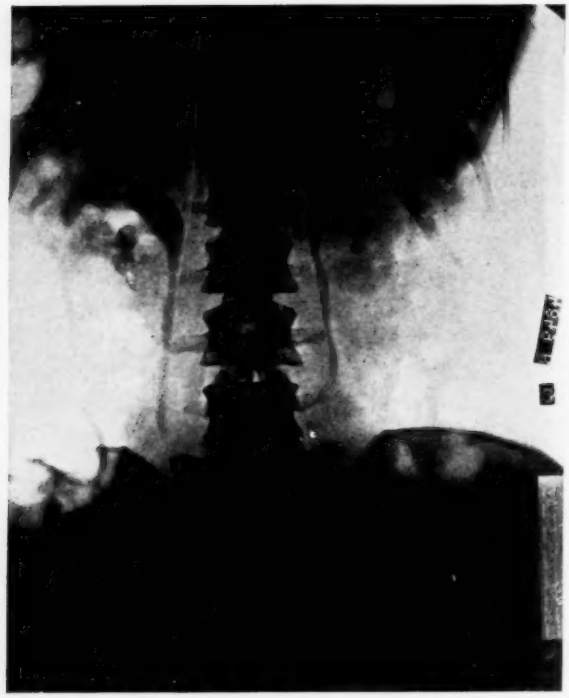

FIG. 2. Female, twenty-eight years old. Tubercular cystitis. Inability to visualize either orifice.

FIG. 3. Male, fifty-five years old. Carcinoma of the bladder associated with a diverticula sac.

FIG. 4.

FIG. 5.

FIGS. 4 & 5. Female, thirty-two years old. Carcinoma of bladder. Inability to visualize either ureteral orifice.

attempt, their work will always remain memorable. It remained for Swick in 1929 to give us a non-toxic drug for intravenous urography, to which was given the name uroselectan, and with it another rung in the ladder of urology has thus been completed.

Fig. 6. Poor functioning left kidney and kinked right ureter; hydronephrosis and hydroureter on right side.

PRESENT METHOD

The purpose of this paper is to emphasize three important points with reference to the method employed with intravenous urography, viz: (1) compression before and during the *x*-ray exposures; (2) increase in the dosage of uroselectan; (3) the withholding of urination.

Although mentioned by the originator in his memorable contribution, greater emphasis seems essential at this time. Notwithstanding the fact that visualization without compression may be quite satisfactory in some clinics, with us at least compression has now become an essential part of this new method. This additional effort is more than compensated for by the advantageous results obtained. The increased density not only of the kidney, but of the entire urinary tract, seems striking in comparison.

Fig. 7. Normal pyeloureterogram.

Compression. The method employed is quite simple. The ordinary Hickey rubber bag is employed as demonstrated in Figure 1, and placed over the bladder region. Enough pressure is used to make the patient just comfortable. This is instituted immediately after the last injection of uroselectan, and allowed to remain until after the first film is taken, then released partly, but retightened within ten minutes before and through the exposure of the second film. The pressure is now entirely released but is reapplied ten minutes before the third, fourth and succeeding exposures, and held compressed during this act. There is no question in our minds that better visualization is obtained by this means, but what the rationale is we are not prepared to say.

Dosage. For the adult Swick now suggests a 60 gm. dose of selectan instead of the original 40 gm. dose. The method of dissolving and distilling is carried out as employed by Swick, viz.: dissolving the

drug in warm double-distilled water to a volume of 110 c.c., filtering this twice through ordinary filter paper, and sterilizing the solution in a water bath at 15 lbs. pressure for one-half hour. With the increased dosage we have found no untoward effects, but much better visualization.

Urination. Although discomforting to some patients, we try not to allow the passage of urine until after the second exposure. If the desire becomes unbearable, by allowing the passage of a small quantity between the first and second exposures, enough comfort may be obtained to carry on without further interference. Compression, no doubt, adds to this extreme desire, so that with slight releasing of the bag, this discomforting feeling may pass.

CONCLUSIONS

1. That all these three points aid materially in producing a denser roentgenogram is without doubt.

2. What the rationale for this improvement may be we are not prepared to say.

3. Increased dosage to 60 gm. has no untoward effects on the patient.

4. External compression over the bladder as here outlined does improve visualization.

5. The value of intravenous urography, as devised by Swick, is tremendous. Only time will evaluate its true worth.

I wish to thank Dr. A. Wolbarst for having made this work possible by supplying the uroselectan employed and Dr. I. Seth Hirsch for his kind cooperation.

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ROENTGENOLOGICAL DIFFERENTIAL DIAGNOSIS OF SPINAL PATHOLOGY*

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THE spine is the most difficult part of the skeletal structure to subject to adequate *x*-ray study. This is due to its complex structure and its anatomical relationship, causing superimposed shadows which interfere with detail. Clear detail is the prime requisite for satisfactory roentgenography.

Improved appliances and added accessories in recent years have made it possible to obtain splendid *x*-ray films in both the antero-posterior and lateral views of the spine. The value of the roentgenogram in the diagnosis of spinal pathology is no longer debatable. The intent of this paper is to show its relative value in different pathological conditions; however, these observations will be confined to the bony structure of the adult spine. Pathological conditions in children are worthy of separate study, while diagnostic measures in diseases of the cord or soft tissues require special technic not within the scope of this paper.

For convenience of consideration, the pathological conditions are divided into 3 classes, as follows:

1. Anomalies and curvatures of the spine.
2. Diseased conditions of the spine.
3. Traumatic injuries to the spine.

Anomalies or congenital malformations may occur in any part of the spine, but certain areas predominate, arranged in numerical value in the following order: The fifth lumbar vertebra, the upper segment of the sacrum, the seventh cervical vertebra, the lower segments of the sacrum, and the first lumbar vertebra. The coccyx normally varies considerably and is not included. Variations in the rest of the spine are exceptional.

The first mentioned, the fifth lumbar, is of unusual interest. Malformations frequently occur and may affect the body, laminae or processes. Occasionally the transverse processes are fused with the sacrum, producing the so-called sacralization of the spine, more often unilateral, rarely bilateral. These processes may also press upon the inner angles of the wing of the ilium; however, an overlapping shadow does not indicate such a condition, as these processes are usually anterior to the ilium with about one-fourth in. clearance. The articular surfaces between the fifth lumbar and the sacrum are often times hazy in the *x*-ray film, due to deposit in this region, not necessarily pathological, but may be painful if inflamed. The irregularity in the development of the two sides of this vertebra frequently causes a slight tipping of the pelvis. Occasionally the fifth lumbar vertebra slips forward at the lumbosacral articulation, a condition called spondylolisthesis. Usually the angulation is increased so that the upper portion of the sacrum may be almost at a right angle to the lumbar spine, while the normal angulation is about 45 degrees. This can be detected with a good lateral film of this region. Sometimes the fifth lumbar is replaced by an extra, or sixth, lumbar vertebra. This portion of the spine is frequently malformed and quite subject to injury. Perhaps it was originally designed by the all-wise Creator for our horizontally-spined ancestors, slowly evoluting toward greater perfection for the comfort of future generations.

The upper segment of the sacrum frequently shows an incomplete fusion of the arch. This in itself is of no pathological significance, but this vertebral segment seems to be more subject to trauma than the normal. The transverse processes of

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the seventh cervical vertebra are occasionally elongated or an extra rib formed. This may be unilateral or bilateral and, if of any length, may produce pain from pressure on the nerves or soft tissues. The lower segments of the sacrum are sometimes incomplete, usually unilaterally. The transverse process of the first lumbar are sometimes ununited, usually unilateral but frequently bilateral, or prolonged into an extra rib. The former condition may be mistaken for a fracture of the transverse process, but should be distinguished by the smooth oval appearance of the articular surfaces. True these conditions may cause or contribute to pathological ailments, but they are very often observed roentgenographically in patients free from symptoms. These defects can only be diagnosed during life by means of the *x*-ray.

The normal contour and degree of motion of the spine can readily be observed by the roentgenologist. The motion between any two vertebrae (except the atlas and axis) is quite limited, although the movement of the spine as a whole is considerable. It can be bent or twisted and if roentgenographed in that position, or if the ray is off center or angulated, the normal spine would appear curved, rotated or deformed. This is worthy of note in medico-legal cases. A fake curvature or rotation can usually be detected by observing the position of certain landmarks. The lower spine should always include the entire pelvis and the symmetry of the spines of the ischae observed. The dorsal spine should include the clavicles and their relative position observed. The structural formation of the bodies of the vertebrae should also be noted.

In a true structural curvature of the spine, there is always rotation of the spine in its vertical axis, with the bodies toward the convexity. Usually this rotation is more pronounced than the curvature. The spinous processes may be in fair alignment; consequently the roentgenogram is the logical way of determining the true degree of deformity. The normal spine can be flexed equally to either side of the center of the spinal plane, while the structural scoliosis cannot be forced to the opposite side beyond this center plane. This can be demonstrated roentgenographically. A kyphosis always shows compression of some of the vertebral bodies over their anterior portion, conforming to the curve. In a lordosis the reverse is true. This condition is easily checked with the *x*-ray film.

Forward or backward slipping of the sacroiliac articulation is hard to confirm. A stereoscopic anteroposterior film, using the Potter-Bucky diaphragm, with vertical shift, and guide wire lines transversely across the film at regular intervals, will give the best perspective. Lateral films of the dorsal spine should be slightly oblique, thus partially avoiding overlapping shadows. Lateral views of the cervical region are best obtained by resting the upper arm against the film, tube centered over the shoulder at 50 inches distance.

DISEASE CONDITIONS

The spine is subject to practically every disease which may affect any portion of the bony structure. Two diseases, arthritis and tuberculosis, stand out preëminently, and coincidently their roentgenographic appearances are practically pathognomonic. In adults, the various forms of arthritis predominate, but tuberculosis easily heads the list when diseased conditions in children are included. Clinically, tuberculosis of the spine is probably the easier to diagnose, especially after the disease has reached the destructive or deforming stage. The body of the vertebra is usually subject to attack, first the anterior portion, then the epiphysis under the cartilaginous discs, with early invasion of these discs, producing a collapse of the body into a wedge shape, with sharp angulation of the spine at point of involvement. Tuberculous disease *per se* is purely destructive, but a subsequent or mixed infection will produce an exudate with

thickening or callus deposit. The roentgenographic appearance is characteristic and scarcely can be mistaken. Arthritis is usually clased as atrophic or hypertrophic, with a severe mixed form extensively involving the spine, hips and other joints, called arthritis deformans. This latter is easily diagnosed clinically; the former is difficult to diagnose clinically, but rather easily diagnosed roentgenologically. The atrophic form usually affects the lower spine and sacroiliac regions, producing a diffuse thinning out of the bone, affecting the articular surfaces, cartilage and ligaments, causing severe pain. The hypertrophic form may affect any portion of the spine; the bodies of the vertebrae are usually involved, with exostoses or spurs projecting from their angles; those from the inferior angle project downward, while those from the superior angle project upward, sometimes called "parrot's beak," due to this peculiar formation. At times, this condition is painful, frequently causing little pain relative to the amount of involvement. The roentgenographic appearance is quite characteristic.

Syphilis offers 3 forms for classification: (1) arthralgia, causing pain without destruction of bone and not recognized roentgenographically; (2) necrosis—destructive process of the bodies and intervertebral discs occurring in the lumbar cervical and dorsal regions, in the order named. Kyphosis may result so the diagnosis from tuberculosis may be difficult. The third is Charcot spine, the result of late syphilis with cerebrospinal degeneration. This produces severe deformity with new bone formation, but causes little pain. This has rather a characteristic appearance.

Malignant diseases of the spine are uncommon. Primary sarcoma is occasionally seen, characterized by rapid destruction of the body of the vertebra, with lack of involvement in the discs. Carcinoma is always metastatic, secondary to this disease in some other region. When observed roentgenographically, the aspect is rather characteristic, showing a diffused, mottled, destructive thinning of the bony structure. This is replaced by tumor mass, so the bodies do not collapse.

Other rare diseases occasionally involving the spine include, osteomyelitis, osteitis deformans (Paget's disease), osteitis fibrosa, hypernephroma, chloroma, progressive myositis ossificans, erosion from an aneurysm, and spondylitis following cerebrospinal meningitis, typhoid, etc., are noted, but they do not offer differential roentgenological signs.

INJURIES

Injuries to the spine, include fractures, dislocations and gun-shot wounds.

Statistics show that 60 per cent of spinal injuries are fracture-dislocations, 20 per cent fractures only, and 20 per cent dislocations alone. It is also stated that 50 per cent of spinal fractures are not diagnosed. This is largely due to the fact that the cord is not involved, the case being diagnosed as a sprain. More universal use of the *x*-ray will undoubtedly lower this large percentage. Fractures of the spine are classified according to the nature of the injury into direct or indirect, those from indirect violence predominating. In these the spine is forceably flexed, causing compression of the bodies, with tearing asunder of the arches and ligaments, while in direct violence the arches are compressed and the bodies separated. In either class the cord is well protected from injury owing to its position near the center of the spine. As stated by Jacobson, "It lies in neutral ground in respect to forces which might cause fracture." It is oftentimes surprising what little damage occurs to the cord in severe injury to the bony framework. In fracture-dislocation injuries from indirect violence the upper segment is usually driven forward on the lower, frequently with partial rotation and some lateral displacement of the body. The first lumbar vertebra suffers more often than any other, due to its location near the center of the spine and at the junction of

the fixed portion of the dorsal spine. Here the leverage would be greatest and this vertebra is slightly smaller than those below. As previously stated, only one-fifth of severe injuries produce dislocation alone. The vertebrae are snugly articulated and securely bound together with ligaments; hence, an injury producing a dislocation would rupture these ligaments and this amount of force usually produces a fracture dislocation. Ordinary strains or sprains do not displace the vertebrae. These bodies are not the nervous, jumpy migratory things some of our modern spineologists would have us believe. In the hundreds of spinal roentgenograms examined I have not seen a displaced vertebra unless caused by severe injury or disease.

Gun-shot wounds differ according to the necessity of removing the foreign body. The *x*-ray is then in order. If the foreign body is not producing pressure symptoms or active infection then it is better to leave it alone. This is our conclusion after seeing a large number of these cases in our work with the Veterans' Bureau.

CONCLUSIONS

The roentgenogram is the only way of diagnosing anomalies, and the best single method for studying spinal curvatures or deformities. A few diseases show pathognomonic *x*-ray signs; in others the roentgenogram is a valuable adjunct in diagnosis.

All injuries of the spine, unless trivial, should be roentgenographed in both the anteroposterior and in the lateral projection.

ROENTGEN LOCALIZATION OF INTRATHORACIC STRUCTURES

WITH REPORT OF FINDINGS IN 19 CASES OF LUNG ABSCESS*

CLAYTON R. JOHNSON, M.D.

LOS ANGELES, CALIF.

STEREOSCOPY and examination of the chest in the lateral position, sometimes combined with lipiodol filling, are roentgen methods commonly used for the intrathoracic localization of normal or abnormal structures. In none of these can any exact information be obtained concerning the location of these structures with relation to anatomical landmarks on the bony thorax.

What I propose here is a simple method whereby the dimensions and location of any structure, whether normal or pathological, may be readily calculated. The method is based on the ordinary stereoscopic roentgenograms made for the diagnosis of suspected intrathoracic pathology.

TECHNIC FOR ROENTGENOGRAPHY

1. Set up the apparatus for stereoscopic roentgenograms of the chest in the upright position.

2. Center the target of the tube to the center of the plate changer with a target-film distance of 180 cm.

3. Arrange for vertical shift of 7.5 cm. each side of the center or a total shift of 15 cm.

4. Place the identification pad bearing the date in the upper left-hand corner of the plate changer. Place the identification pad bearing the patient's number in the upper right-hand corner of the plate changer.

5. Make stereoroentgenograms with suitable exposure during a single period of inspiration.

This set up is used in all examinations of the chest. The localization of any unusual structure may then be made a part of the first report.

TECHNIC FOR CALCULATION

1. Place the stereoroentgenograms in the stereoscope, and on one of the films place ink dots on the rib anteriorly and the rib posteriorly at points which lie in a horizontal line with the structure for localization.

2. Superimpose the two films in such a way that the shadows of the markers in the upper corners of the roentgenograms are exactly superimposed and fasten the films together with spring paper clips. Note if the right-hand edges of the films are exactly superimposed and parallel. If they are not, then draw a line marking the edge of the overlapping film. This procedure is to insure a parallel shift in marking the points for calculation.

3. Remove the paper clips and shift the films over one another in a longitudinal direction, keeping the right borders of the films or the border as marked in paragraph 2 parallel and superimposed, until the shadows of the anterior rib marked in paragraph 1 are exactly superimposed. With a needle perforate both films at the point marked in paragraph 1.

TABLE 1

Case	Age	Sex	Dur.	Size	Depth	Loc.	FML	Conf.
I	46	M	3 mo.	3.0 x 1.5	2.0	7LP	8.0	Op.
II	40	M	2 yr.	5.0 x 2.5	2.0	3RA	10.0	Op.
III	29	M	6 wk.	2.5 x 2.5	1.5	2RA	8.0	
IV	16	F	1 yr.	2.5 x 2.5	2.5	8LP	8.0	
V	53	M	6 wk.	2.5 x 2.5	4.0	5RP	8.0	Aut.
VI	31	F	1 yr.	2.0 x 2.5	2.0	8LP	6.0	Op.
VII	70	M	.....	3.5 x 3.0	4.5	4RA	10.0	
VIII	42	M	2 mo.	4.5 x 4.5	2.0	10RP	5.0	Aut.
IX	27	M	3 wk.	4.0 x 2.0	2.0	5RP	8.0	
X	23	M	.....	3.0 x 3.0	3.0	8RP	8.0	
XI	27	F	1 yr.	2.0 x 1.5	3.0	6RP	8.0	
XII	68	M	2 mo.	3.0 x 2.5	2.0	3RA	8.0	Op.
XIII	71	M	2 mo.	2.5 x 5.5	1.0	9RP	8.0	
XIV	37	M	.....	2.0 x 2.0	3.0	7LP	9.0	Op.
XV	40	F	2 mo.	2.0 x 2.5	2.0	2LA	6.0	
XVI	62	M	.....	3.5 x 5.5	4.0	7RP	7.0	Aut.
XVII	31	M	3 wk.	3.0 x 3.0	3.0	6LP	6.0	
XVIII	45	F	3 yr.	3.0 x 3.0	2.0	4RA	8.0	Op.
XIX	30	M	1 yr.	4.0 x 4.0	2.0	2LA	4.0	Op.

Abbreviations: Dur.—duration; Loc.—location; e.g. 7LP—7th rib, left posterior; FML—from midline: Conf.—confirmation.

* Read before the regular monthly meeting of the Trudeau Society, Los Angeles, March 25, 1930.

4. In like manner superimpose the shadows of the posterior rib and perforate both films at point marked in paragraph 1.

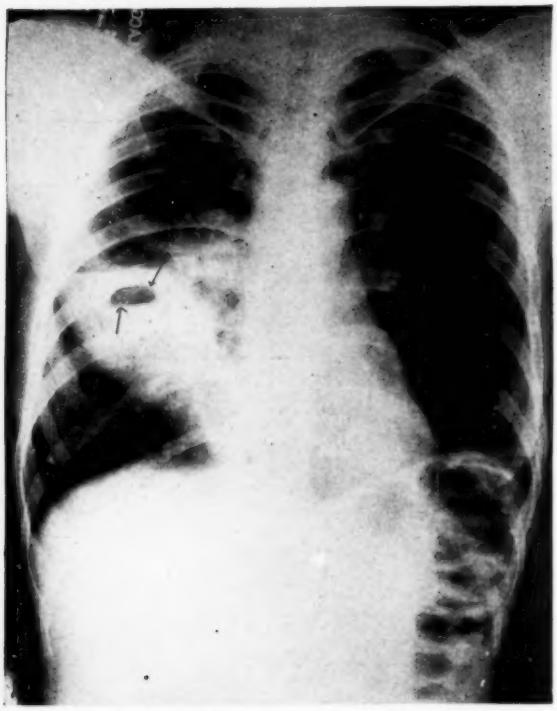

FIG. 1. Case XVIII. Abscess cavity 3 cm. in diameter. Center 2 cm. posterior to point on upper border of right fourth rib, 8 cm. to right of midsternal line.

5. Superimpose the shadows of the structure for localization, still keeping the right hand borders of the films superimposed and parallel, and clip the films together.

6. Measure the distance in millimeters between the points marked on the anterior rib. This distance times 10 will be the depth of the structure as measured from the point selected on the rib. For example in Case XVIII, Figure 1, when the shadows of the abscess cavity are superimposed the distance between the points marked on the anterior rib is 2 mm. The product of 2 and 10 is 20 mm. Therefore the approximate center of the cavity is 20 mm. or 2 cm. from the point marked on the rib. In like manner the distance may be calculated from the point selected on the posterior rib.

7. Measure the distance in centimeters to the right or left of the midsternal line if the structure is located anteriorly, from the midvertebral line if located posteriorly. The plane dimensions of the structure may be measured directly from the radiogram.

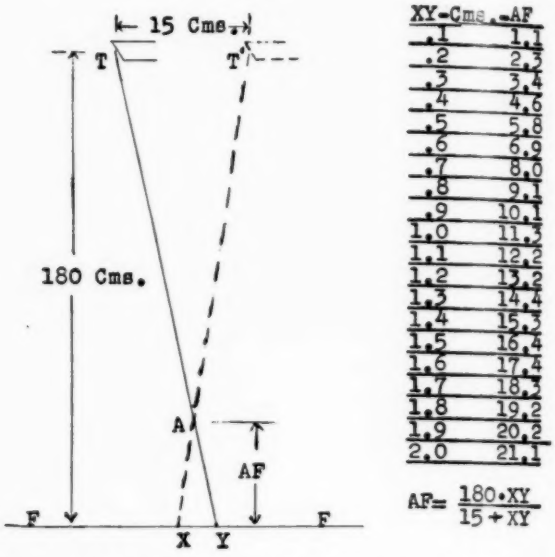

FIG. 2. Relationship of displacement of shadow of point to distance from film, according to formula of Buguet and Gascar. (See text.)

A representative report should then read as follows:

Case XVIII, Figure 1: There is an abscess cavity in the right lung. Its diameter is 3 cm. Its center is 2 cm. posterior to the upper border of the right fourth rib at a point 8 cm. from the midsternal line.

An abscess cavity is roughly spherical or ovoid in shape. Its shadow is cast by those rays tangent to its lateral surfaces. Its approximate center will then lie in the vertical plane of the margins which cast the shadow. The surface of the cavity nearest the chest wall will be closer to the chest wall than the center of the cavity by one-half the shortest diameter of the cavity. In the preceding instance, with a cavity 3 cm. in diameter with its center 2 cm. from the chest wall, the near point of the cavity should be within 0.5 cm. of the chest wall. In practice this has been proved to be approximately correct.

DISCUSSION OF THE METHOD

For all practical purposes, a roentgenogram of structures at a maximum distance

of 8 in. from the film taken with a target-film distance of 72 in. (180 cm.) will have a maximum distortion of 10 per cent. The closer the object to the film the less the distortion. Therefore it is possible to measure directly from the film dimensions of length and breadth.

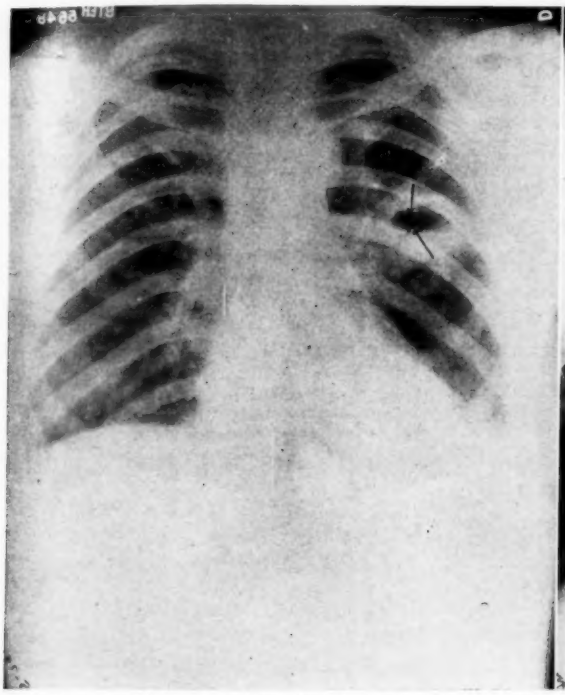

Fig. 3. Case I. Abscess cavity in left lung. Size 3 cm. by 1.5 cm. Its center 2 cm. anterior to a point on the left seventh rib, 8 cm. to the left of the midvertebral line.

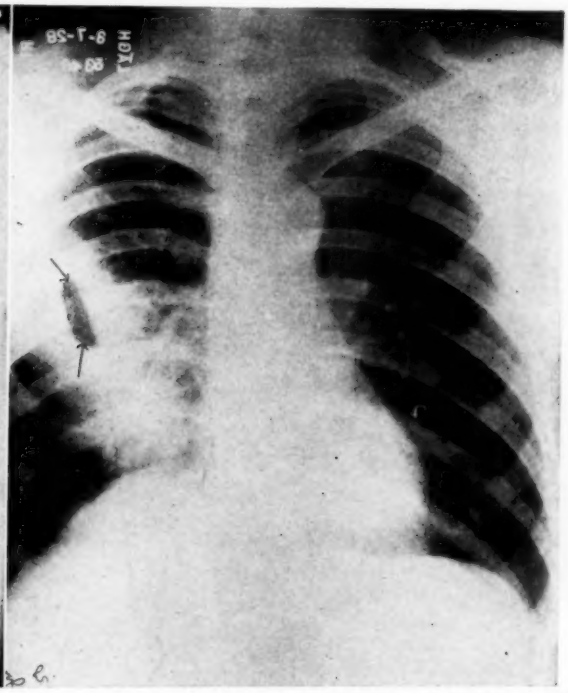

Fig. 4. Case II. Abscess cavity in right lung. Size 5 cm. x 2.5 cm. Center 2 cm. posterior to a point on right third rib, 10 cm. to right of midsternal line.

The shift of the shadow of the structure on stereoscopic roentgenograms determines its distance from the film at the time of the exposure according to the diagram, table and formula* of Figure 2. AF is the unknown distance of the object from its roentgenogram; 180 cm. is the target-film distance; 15 cm. is the shift of the target; XY can be measured directly when the stereoscopic roentgenograms are superimposed. By substituting varying values for XY from 0.1 to 2.0 cm., the values for AF were calculated as shown in the table.

For all practical purposes an increase in XY of 0.1 cm. causes an increase of AF of 1.0 cm. The vertical distance between the depths of any two points is the difference between their distances from their roentgenogram. For example, in Case XVIII a point on the right fourth rib anteriorly has a shift in its shadow of 3.0 mm. (0.3 cm.). The margin of the abscess cavity has a shift in its shadow of 5.0 mm. (0.5 cm.). The difference between the shift of the rib and the shift of the cavity is 2 mm. Therefore, the margin of the cavity must be 20 mm. or 2.0 cm. from the rib. If we wish to be more exact, the values of AF may be found in the table. In this particular instance the depth of the cavity would be the difference between 5.8 cm. and 3.4 cm. or 2.4 cm.

To further simplify the procedure one needs simply to superimpose the shadows of the structure and measure the shift in the shadow of the adjacent rib. This distance in millimeters multipled by 10 will be the approximate depth of the structure.

REPORT OF FINDINGS IN 19 CASES OF SIMPLE LUNG ABSCESS

During the past year this system of

* Formula of Buguet and Gascar, 1896.

localization has been used in 19 cases of simple lung abscess. Seven of the cases were treated surgically by Dr. H. E. Schiffbauer, Senior Attending Surgeon on the Chest Surgical Service at the Los Angeles General Hospital. The exact location of the abscess cavity in all of these cases was successfully calculated before operation. The location of four more were confirmed by autopsy. The accompanying table (Fig. 3) gives a summary of the findings in all cases.

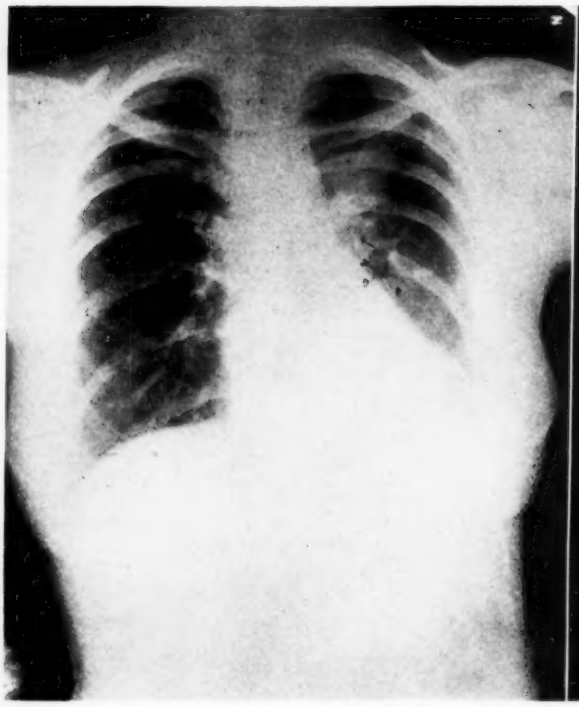

FIG. 5. Case VI. Abscess cavity in left lung. Size 2 cm. x 2.5 cm. Center 2 cm. anterior to point on left eighth rib, 6 cm. to left of midvertebral line.

FIG. 6. Odd-shaped bullet in lower left thorax. Length 3.5 cm. Location 7 cm. posterior to lower border of fourth rib, 10 cm. anterior to lower border of eighth rib, and 3 cm. to midline of sixth rib in midaxillary line.

On reviewing these findings it should be noted that in the majority of instances the cavity was subpleural. It was located either anteriorly or posteriorly in the thorax. In most cases the cavity was accessible to surgical drainage. We have encountered one type in which surgical approach was somewhat complicated. This type is that in which the cavity occurs along the distribution of the posterior upper lobe bronchus and lies near the interlobar fissure. In this instance the upper portion of the lower lobe may lie between the chest wall and the abscess cavity. This occurred in Case XIV.

CONCLUSIONS

Intrathoracic localization may be made a comparatively simple procedure readily applied by the experienced roentgenologist. It is applicable to all those instances where accurate localization of intrathoracic structures may be of value in diagnosis or treatment.

A report of a roentgen examination of the chest is not complete without giving exact information as to size and location of any unusual structure.

This method has been found particularly applicable to the localization of simple nontuberculous lung abscess cavities, to the localization of foreign bodies within the thorax and to the localization of certain tuberculous lesions.

RADIATION THERAPY FOR BENIGN UTERINE HEMORRHAGE*

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THE title of this paper has been chosen significantly, after the elimination of conditions not included in this catagory.

While the cause of menorrhagia or metrorrhagia was, as a rule, recognized to be a fibromyoma, a number of bleeding patients presented a rather obscure etiology. Six out of the number reviewed were married women between the ages of twenty-five and thirty-six. The diagnosis of fibrosis uteri was suggested tentatively, although the author doubted that this etiology applied to all of this series.

Schmitz[1] presented a logical classification of the types of benign uterine hemorrhages as follows: (a) hyperemia; (b) polymenorrhea; (c) metrorrhagia. The difficulty of differentiating among the three types, however, is evident.

The diagnostic curettage so much heralded by gynecologists, while a necessary prerequisite to radiation therapy, is not always *the* possible procedure in private practice. Most patients consider this a surgical risk and refuse to submit to it.

Stacy and Mussey[2] limit their curettement to patients more than forty years of age. At any rate, its importance prior to the insertion of *radium* is obvious.

Thirty-four patients who gave definite physical signs of tumor varied between the ages of thirty and fifty-five. The number of masses felt in the pelvis varied from one small nodule in some, to several large tumors in a few patients. One patient in this series in whom the result was only palliative, had an enormously enlarged uterus, and although the contraindications on account of the doubtful result were explained to the patient, she absolutely refused an operation. She returned recently after a period of six months of freedom from symptoms, with recurrent bleeding. Another patient in whom the result was poor, proved eventually to have had a benign intraligamentous tumor.

The indications and contraindications were naturally considered. The indications in the majority of patients of this series corresponded with those promulgated by J. C. Mason:[3] (1) past normal age of menopause; (2) history of menorrhagia; (3) tumor less than 15 cm. in diameter; (4) fibrous type uterus; (5) essential uterine hemorrhage; (6) young patients who refuse operation; (7) bad surgical risks.

The chief objective symptom in the author's series was hemorrhage. Subjectively, backache and "bearing-down pain" were the complaints with larger tumors.

The hemoglobin estimation and blood count in a number of patients were always below normal, while in several, the hemoglobin was less than 70 per cent and the erythrocytes fewer than 3,500,000. The hematopoietic regeneration invariably occurred with the cessation of the bleeding.

Since Crile's[4] utterance in 1924 that "in the child-bearing period, radium and *x*-rays should be used only in the treatment of cancer of the cervix, since surgery relieves practically all cases and preserves the ovarian balance," many investigators, both radiologists and gynecologists, have amply demonstrated the possibility of preserving the conceptive powers of the adnexa exposed to radiation.

While the regional classification was as a rule attempted, the uncertainty of accuracy readily suggests itself, in view of the fact that a mass felt subserously may be wholly intramural where most of the fibromyomata originate. Furthermore, the presence of a subserous mass does not eliminate the possibility of a submucous nodule. Although the clinical results are

* Submitted for publication April 2, 1930.

uniformly good, the regional classification is also important in indicating the proper type of emanator of radiant energy. The *submucous* fibromyomata or *pedunculated* tumors are likely to ulcerate on account of the too intense *radium* bombardment. Better results may be expected with *x-rays*, although pedunculated tumors should be removed *surgically*. The large *subserous* fibromyoma does not react so well with *radium*. The reason for this may be explained as follows: the radium applicator being inserted in the uterus is a goodly distance from the mass, especially if the latter is centripetally situated. Furthermore, the intensity of radiation varies as the square of the distance, hence an insufficient amount of rays reaches the subserous neoplasm. However, radium is the applicator of choice in young women suffering from idiopathic uterine hemorrhage because the dose to be applied can be controlled more accurately. Clinical experience has taught us that 250 to 800 milligram or millicurie hours will produce a temporary amenorrhea which may last two to six months. The castration dose is two to three times greater.

In the author's series radium was applied eight times with invariably good results. In only 1 of the 6 (aged thirty-five) hemorrhage cases a permanent amenorrhea was produced with the *x*-rays. In another of this series, the patient became pregnant some months later. Phahler[5] cited his unusual experience with a patient in whom a uterine fibroid disappeared with irradiation without a temporary amenorrhea. Subsequently she gave birth to two healthy children.

Although the author has by choice employed the *x*-rays, he did not hesitate to use radium where indicated and the patient's financial status permitted it. The cost of radium and the necessary hospitalization are an item which is a serious consideration to the patient of moderate means.

At present the author employes deep penetrating *x-rays* of 180 kv. peak and filtered by 0.5 mm. of copper and 1.0 mm. of aluminum. The target to skin distance is 50 cm. Fractional exposures twice weekly are given until a full erythema dose is administered. This is usually completed within three weeks, depending upon the urgency. Other series are repeated within six weeks as indicated. Some tanning and an occasional bronzing of the skin occur.

In most patients radiation is given through one portal, 15 cm. in diameter in the suprapubic region, and another through the sacral region. In some patients with multiple tumors, irradiation is done through a portal on either side of the median abdominal raphe.

The irradiation with *radium* involves hospitalization of the patient, meticulous preparation of the genitalia and the patient as for major surgery; the properly prepared radium salt or emanation is then inserted into the uterine cavity. The dose given varies from 250 to 2000 mg. hours or its equivalent in millicuries.

RÉSUMÉ

1. Radiation therapy is an excellent remedy for benign uterine hemorrhage due to fibromyomata or the type classed as idiopathic, with few exceptions.

2. X-ray therapy is superior for all types of fibromyomata and it is also less expensive for the patient of moderate means.

3. Radium is an excellent remedy in selected cases, especially in young women, but more expensive.

4. Finally, radiation therapy is a potent but a dangerous remedy in the hands of the tyro. Improper irradiation unwarrantably discredits a fine therapeutic agent.

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A PRACTICAL X-RAY SERIALOGRAPH*

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STARBUCK, MINN.

THE writer wishes to present herewith an *x*-ray accessory for making serial roentgenograms, and a method of suspending it in a convenient manner in a permanent installation alongside the fluoroscope.

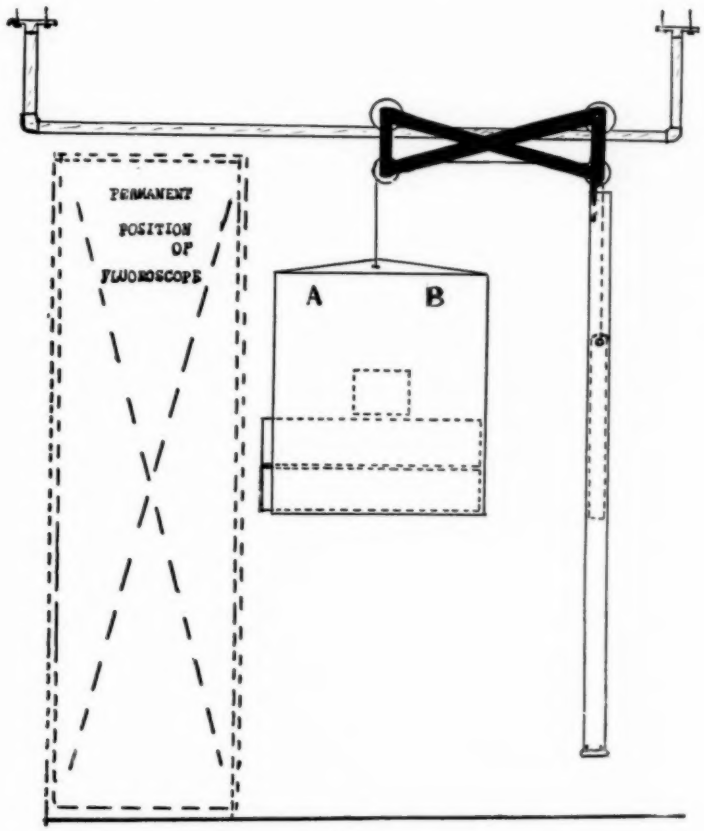

FIG. 1.

The tunnel is made to accommodate one regular 10 x 12 in. cassette, and produces on this film six separate roentgenograms, each taken a short interval after the preceding one. This tunnel is 20 x 24 in. outside measurement and weighs about 32 lb. It is made up in the regular manner with a sheet of 1 mm. of aluminum next to the patient, then a sheet of lead 2 mm. thick with the 4 x 5 in. aperture in its center; then the proper space for the cassette, and lastly the backing of wood and bakelite to give it strength and rigidity. Through an opening near the bottom of one side are inserted two strips of wood 4 in. wide and 22 in. long.

In operation, the tunnel is pulled along on its trolley and then lowered between the patient and the fluoroscopic screen; the portion desired is then centered in the 4 x 5 in. opening, and the patient by grasping the edges of tunnel and fluoroscopic screen holds all tightly against his abdomen. The 10 x 12 in. cassette is then inserted in the space at top and brought over to position "A" and an exposure made, then the cassette is moved over to position "B" and the second exposure

* Submitted for publication May 23, 1930.

made. The upper of the two wood strips is then pulled out, allowing the cassette to drop down 4 in., and two more exposures are made in a similar manner, and then the bottom board is pulled out and the last two exposures made. In this way six exposures are quickly made, each 4 x 5 in., on one 10 x 12 in. film, and all six are right side up instead of two right and two upside down as with the original model on which this idea is an improvement.

The trolley system consists of 1 in. gas pipe fastened by floor plates to joists in the ceiling by lag bolts. This is constructed and placed in such a manner that no portion comes nearer to the overhead high tension wires than the original construction of the fluoroscope involves, passing entirely under and around the wires in cases where they approach the fluorscope from the front. The trolley wheels are turned to fit the pipe and by bracing as shown a very light yet rigid construction is achieved. The counterbalancing system consists of the nickel-plated tubular standard of an old *x*-ray tube stand and its original weight sliding within it. The tunnel and weight are easily equalized by melting out or adding to the required amount of lead.

When not in use, the entire apparatus is easily shoved over to one side on the trolley where it remains always ready for instantaneous use.

For handling the cassettes when within the tunnel, one may bore a hole near the corners at one end into which are fitted the hooked ends of a wire bail. This does not injure the cassette, is more convenient than a pan, and does not alter the cassette so as to prevent its use in the ordinary manner.

THE ACHIEVEMENTS OF UTEROSALPINGOGRAPHY*

JOSEPH KALDOR, M.D.

BROOKLYN, N. Y.

IN THE year 1914 Dartiques and Dimier by their "Radiographie Periuterine et Endouterine" introduced for the first time the new method of uterosalpingography. Independently, Cary of Brooklyn and Rubin of New York advanced the identical method in 1914. Regarding priority, the credit can be given to Cary and Rubin as Dartiques and Dimier did not give their results publicity until 1916.

Since then uterosalpingography, or as some prefer to call it hysterosalpingography, has proved almost indispensable in certain phases of diagnostic gynecology and obstetrics, and has revealed new facts in the anatomy and physiology of the female genital organs.

The success of uterosalpingography depended entirely upon the use of a proper medium which had not only to give a strong *x*-ray shadow, but at the same time avoid any harmful action on the tissues. Dartiques and Dimier used 7 to 8 c.c. of a 10 per cent collargol solution for the visualization of the uterus and adnexa. Later on argyrol and bismuth paste were used by others. But these solutions did not give satisfactory *x*-ray shadows and furthermore caused untoward effects, such as peritonitis, abscess fomation and adhesions. It was due to the failure to find a proper agent that uterosalpingography was not generally applied as a diagnostic method until about 1923.

In the interim Zimmerman used umbrenal; but this had a very bad effect on the mucous membrane of the tube and on the peritoneum of the Douglas pouch and therefore failed to gain general acceptance.

In the search for satisfactory contrast material many men deserve credit. For the sake of brevity we shall only mention the following few: Mocquot, Tussau, Schneider, Jarochka, Arnstam, Serdukoff, Carelli, Randall, Kennedy, rather an international galaxy and showing the study to be of widespread interest.

Among the agents most widely used were bromnatrium, collargol, argyrol, sodium iodide, iodakol, iodipin and lipiodol. At first the solutions containing iodine were used with care and trepidation because of the fear that they might have a destructive action on the tubal epithelium or show toxic effects. But experience showed that these considerations were not warranted.

Among the iodized oils, iodipin and lipiodol gained foremost reputation due to their merits and recently the latter has almost unanimously been given first choice. Iodipin was first used by Winternitz. One cubic centimeter of iodipin contains 0.51 gm. iodine in an organic combination. In the opinion of Zimmerman and Nahmmacher it is harmless to the ciliated epithelium of tubal mucosa and its only drawback is its heavy viscosity. But Dyroff claims it to be decidedly harmful because it causes oily deposits of long duration, fibrinous exudates and adhesions.

Lipiodol was used by Lafay in 1901 for therapeutic purposes only and as contrast material in 1925 by Sicard and Forestier. One cubic centimeter of lipiodol contains 0.54 grams of iodine in a vegetable oil combination and is tolerated by the organs with which it comes in contact as the least irritant of all known solutions and gives the sharpest *x*-ray shadow.

Preliminary to uterosalpingography the patient should receive a bowel evacuant the night before, and be put on the *x*-ray table with an empty bladder. In order to gain better visualization, Saint Portret

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and Dimier recommend rectal and vesical insufflation; Heuser, Carelli, O'Campo and Arens favor a pneumoperitoneum in conjunction with the uterosalpingography, but it may be safely said that no special advantages can be gained by the use of this combined procedure.

The instrumentarium is simple and easy to handle. It consists of a vaginal speculum, a tenaculum, a syringe with long nozzle and the contrast material. Some prefer to introduce the contrast material by means of a catheter, others use the rigid nozzle. Some have a special pressure-reading attachment on their syringe in order to control the applied pressure, as in the "Schneider-Beclere Hysteromanometre." Others use a different sized nozzle, still others apply a rubber stopper on the nozzle to prevent the backflow of the injected solution, and some use both. Temesvary attaches his syringe to the tenaculum in such manner that the nozzle is held firmly in the cervix and at the same time the cervix is pulled down. In this way the injection can be managed with one hand. An inexpensive and yet very efficient one is a modified Ultzman-Record syringe.

The appropriate time to do uterosalpingography is midtime between menses. Shortly before or after menstruation the swelling of the endometrium may occlude the uterine ostia of the tubes and prevent the contrast material from entering into them.

The instruments used must be sterile and the procedure executed under strict precautions of asepsis. The technic is as follows:

The patient is put on the *x*-ray table, recumbent, or as some prefer, in the stone-cutting or Trendelenburg position. The vaginal speculum is inserted, the vagina and cervix thoroughly disinfected and after the cervix is grasped, the nozzle of the syringe is inserted into the canal and the lipiodol injected slowly, about 1 c.c. per minute. Those who use the manometer, control the applied pressure by the reading; but their recommendations vary from 60 to 200 mm. Hg pressure, sufficient proof that this precaution is unnecessary. The best manometer after all is the hand, which will recognize readily any undue resistance and will not assert itself against it. As to the quantity of the injected fluid, no definite ruling can be set: it varies with the individual case.

Some precede the radiography by fluoroscopy. Fluoroscopy will give information about the degree of filling and the contractions of the uterus; but as to finer details, only the radiography is satisfactory.

If only information about the uterus is sought for, the radiography may follow immediately upon the injection; but if the tubes have to show up on the plate, it is safer to wait from eight to ten minutes. A second and third film should be exposed one hour and three hours after the first picture, but without repeated injections. These radiographs are made for the sake of control and for further information about the localization of the contrast matter.

In case the tubes are patent and the lipiodol reaches the abdominal cavity, it will undergo absorption there. As to the length of time that this will require, the opinions vary: some believe a few days, others claim that it takes weeks or months. When found at any later date in the abdomen it never seems to have caused any irritation of the peritoneum and in Schober's examinations these lipiodol rests were always sterile.

Gregoire and Darbois employ lumbal anesthesia but this is not necessary because uterosalpingography hardly ever causes any pain beyond discomfort of short duration. In fact it is tolerated much better than gas insufflation.

In regard to the anatomy and physiology of the female genital organs, uterosalpingography has revealed the following facts:

The position of the uterus is rarely in the midline of the pelvis; but I myself am inclined to believe that this deviation from the pelvic axis is due to the traction exerted upon the cervix during manipulation. The cavity of the normal uterus is always

triangular. This triangular shadow varies in size on the *x*-ray film and is dependent on the position and version of the uterus. The more stretched the uterus is, the higher will we find this triangle to be: retroversion without retroflexion enlarges the shadow cast upon the film, and the more pronounced the retroflexion, the shorter it will measure.

At the fundus from cornu to cornu, this shadow measures from 3.6 to 4.3 cm. in normal uteri. From fundus to cervix, the measurement varies; but if it is over 4 to 4.5 cm., it is in direct ratio to the retroversion.

The uterine cavity fills up with 3 to 10 c.c. of lipiodol in normal conditions, dependent on the tonicity of the musculature and the applied pressure.

Reinberg and Arnstam observed under the fluoroscope that after filling the uterus by way of a catheter, in some cases part of the contrast material was at once expelled back into the vagina, and part of it was forced through the tubes into the abdomen. From this they concluded that there is not only an expelling muscular action of the uterus, but also a peristalsis on the tubes from cornua to fimbriated end. It is known that the normal peristalsis is from fimbriated end to cornua; but that there is also an antiperistalsis is proved by Henkel, who injected only 0.5 c.c. of iodipin into the uterus, placed the patient in Trendelenburg position, and on radiography half an hour later, found the iodipin in the tubes. Temesvary and Mikulicz believe that antiperistalsis takes place only if the backflow of the contrast material meets obstruction in the cervix. They introduced into the uterine cavity a lipiodol saturated applicator and upon withdrawing this quickly found that the expulsion of the lipiodol took place towards the vagina. However, if they left it in place so as to occlude the internal os, the lipiodol was observed to ascend into the tubes. On the *x*-ray film the peristalsis of the tubes can be recognized by the fact that the contrast material does not give a continuous shadow, but that it is broken up into fractions.

In 1926 Heuser from his observations concluded that there is a salpingouterine sphincter action and later Romcke proved this physiological supposition by his anatomical findings. This sphincter is situated about the middle of the intramural section of the tube. Its presence was proved by Schneider and Eisler on the basis of manometer readings. On the removed organ this sphincter is tetanic enough so as not to allow the filling of the tubes from the cervix, whereas in the living organ, the same specimen showed easy permeability. In the living, due to some unknown reason, the sphincter may be in a spastic stage and, in order to differentiate between such state and an organic, permanent obstruction at the sphincter's site, atropine should be given to the patient ten minutes prior to the injection of the lipiodol which will bring about a relaxation if the closure is only functional.

The tube is capable of elongating to an unbelievable length, as is seen in cases of prolapsed uteri where the cervix protrudes beyond the labia and the tubes remain in the pelvis and reach far up.

Further facts revealed by uterosalpingography and reported by Susaki are:

Thirty days after birth the uterus is still not normal in size. The cervix assumes its prenatal form in multiparas much sooner than in primiparas and in the early puerperium we find the uterus mostly in an anteposition and anteversion. In the late puerperium it is mostly in retroversion.

Besides these ascertained anatomical and physiological factors, uterosalpingography offers more valuable service in diagnosis. Its foremost application is in cases of sterility. According to Jarcho, "it not only supplements but frequently supplants insufflation of gas." If the Rubin test shows the tubes to be patent, reasonable doubt can be maintained whether one or both tubes are permeable, and if the manometer reading indicates obstruction, information as to the site of the trouble is

still missing. These drawbacks are eliminated by the properly executed uterosalpingography. It gives us information about both tubes and by localizing the site of lesion will indicate operative possibilities for the cure of sterility. Furthermore the iodized oil may have definite therapeutic advantages in subacute and chronic conditions of the Fallopian tubes.

If the tubes are patent we will find on the *x*-ray film shadows of droplets and clots of lipiodol in the free abdominal or pelvic cavity. If the tube shows up as possessing a blunt end, like a club, one must not conclude from this appearance that the fimbriated end is closed; there is a possibility that the lipiodol is filming it and gives the impression of obstruction. In such doubtful condition it is advisable to follow up with another radiograph an hour or two later, to search for the only conclusive signs of tubal patency, i.e. droplets and clots of lipiodol or to repeat the whole procedure by applying more pressure during the injection.

Henkels and Rouves base their diagnosis of ovarian tumor on the fact that the tubes appear extraordinarily elongated. The diagnostic point of Rosenblatt and Kesz is, that the surfaces of these growths become covered with lipiodol and show up as some part of a spherical body. According to others the diagnosis is made on the basis of negative *x*-ray findings, i.e., the tubes and uterus are normal but the clinical findings point to some growth.

In differentiation of myomas it is fairly satisfactorily established that in submucous myomas we will find a filling defect in a deformed uterus. In intramural myomas, instead of the triangular uterine shadow, we will find an asymmetrical and deformed one. We are to note that a submucous myoma of the posterior wall will show up only if the film is exposed in a lateral position. Incidentally I wish to mention that Samuel noticed, upon using a 25–30 per cent sodium bromide solution as contrast material in the visualization of myomatous uteri, that as a sequel these myomas decreased essentially in size and that the menorrhagia diminished. It is explained by the dehydrating action of this highly concentrated and hygroscopic salt solution.

The diagnostic signs of a four to six weeks' pregnancy are according to Popovitc the following:

1. Form changes, i.e. the uterus is larger, symmetrical and atonic.
2. Under the fluoroscope irregular contractions are observed.
3. These contractions become regular, almost rhythmical, under the influence of pituitrin.
4. A filling defect is seen in some part of the uterus corresponding to the location of the ovum. This defect is always convex, broader than long, and the edges are never clean-cut, in differentiation from a submucous myoma.

In missed or incomplete abortion all these signs may be detected except the last mentioned one. The filling defect is not present and instead, according to the fact that the contrast material has mixed rather unevenly between the different and loose parts of the products of conception, the picture is that of a spherical bag filled with different sized and shaped fragments of lipiodol shadows.

In spite of the fact that many men, as Carlos Heuser, Dyroff and Arnstam have applied uterosalpingography to determine a question of pregnancy and have claimed that it is harmless to the mother and that it does not bring about an abortion, Joachimovitz found that iodine absorption takes place readily in pregnant women, causing severe acne eruption. Many others would not apply this diagnostic method for fear of harming the fetus. We are inclined to take sides with the latter and esteem pregnancy as one of the contraindications to uterosalpingography.

In the diagnosis of tubal pregnancy the following points may be utilized:

1. The gravid tube either does not fill with contrast material at all or fills up only very slowly and to an insufficient degree.

2. The isthmic portion of the tube is larger than usual.

3. In the pregnant tube the distal end of the contrast material shadow stops suddenly and is concave towards the fimbriated end. This phenomenon is explained by the fact that both the lipiodol and the tubal contents as a whole are elastic bodies and on the surface of their contact the tubal contents will resist the pressure of the lipiodol, causing it to stop in a concave line.

4. Sometimes the gravidity filling defect can be seen in the tube.

5. The previously mentioned signs of pregnancy are present on the uterus itself.

It must be remembered that in tubal pregnancy uterosalpingography involves some danger through the possibility of causing a rupture.

Other uses of this method are in the diagnoses of malformations, in the differential diagnosis between uterine and adnexal tumors and in certain postoperative follow-up cases.

As contraindications, the following are generally accepted: fever, gonorrhea, pregnancy, malignancy and inflammations, although Cotte and Bertrand apply it even in such cases for therapeutic purposes.

Before closing this review I want to call attention to a perversion to which uterosalpingography has led some men. They have injected 15 to 20 c.c. of lipiodol by way of a Douglas puncture in order to reveal diagnostic facts in cases of tumors and adhesions. It is needless to say how hazardous and unnecessary is such an undertaking.

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DISPLACEMENTS OF THE UTERUS*

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ONE must at least briefly review the normal supports of the uterus before entering upon the discussion of the subject of uterine displacements. Usually we find that the displacements follow upon damaged or diseased supports. Under normal conditions the tone of the supportive structures is sufficient to maintain the uterus in good position.

The uterine supports are loosely arranged in order that the uterus may undergo considerable mobility. The pregnant uterus gradually changes its position from the beginning to the end of pregnancy. This marked alteration in position and size of the uterus could only occur with loosely arranged attachments. The degree of fullness of the bladder alters the position of the uterus. The normal degree of uterine antiflexion can only occur when the bladder is empty. A distended lower bowel tends to push the cervix forwards and this will carry the uterus backwards. Physiologically the uterus will be in its most normal position when the bladder and rectum are empty or nearly so.

The peritoneal ligaments are morphologically of the nature of a mesentery. They carry the nerves and blood vessels to the organ and offer resistance only to gross displacements, leaving the uterus a wide range of mobility. The mobility of these attachments allows of the uterus being drawn forwards and upwards to or through the abdominal wound during operation. These ligaments therefore are not normally true supports of the uterus. The round ligaments compare morphologically to the gubernaculum testis of the male. The function of this structure is developmental only and the portion which becomes the round ligament may be regarded as a vestigial structure which serves no definite purpose in the adult. Its normal laxity and its marked variation in size seem to obviously mean that it has but little to do with holding the uterus in its normal position. These ligaments are serviceable however to the surgeon when he wishes to use them as supportive structures.

The uterus is held in position mainly by the fascial and muscular structures which also serve to close over the lower part of the pelvis. These structures may be conveniently arranged into two groups, (a) the superficial and (b) the deep. The superficial or sphincter group is made up of: the superficial sphincter ani; the transverse perenei; and the sphincter vaginae muscles; along with the superficial and deep layers of perineal fascia. The deep group constitutes the pelvic diaphragm. It is made up of the levator ani and the coccygei muscles, along with the visceral layer of pelvic fascia. This pelvic floor is weakened by the passage through it of the vagina and the urethra. This element is however partly counterbalanced by the fact that the vagina and urethra are attached to the sloping sides of the pelvic floor by a layer of pelvic fascia which is continuous with the general pelvic fascia and attached to the pelvic wall at the "white line." Another assisting factor is that the uterine vessels bring with them an investment of fibrous tissue. The so-called transverse cervical ligament adds further support. The definite support offered by this fibrous tissue around the lower uterine segment and upper part of cervix is appreciated in doing a vaginal hysterectomy. It is difficult to pull the cervix down to a lower level until these lateral attachments have been severed. When the uterine vessels and transverse cervical attachments have been severed the cervix can be readily pulled downwards,

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as the upper peritoneal attachments allow so much mobility that the uterus may be readily delivered and freed from those attachments. The uterus is therefore maintained at its normal level chiefly by the following supports: (1) pelvic diaphragm, (2) transverse fascial and fibrous bands, (3) fibrous sheaths of blood vessels.

The displacements of the uterus may be considered as follows:

(a) Forward displacement: anteversion, anteflexion.

(b) Backward displacement: retroversion, retroflexion.

(c) Downward displacement: prolapse.

(d) Inversion: turning inside out.

Forward Displacement: Anteversion is the normal position of the uterus. The degree of anteversion will vary with the filling and emptying of the bladder.

Anteflexion may be congenital or acquired. The congenital variety is rare. It is often associated with some degree of under development of the uterus. The functioning of the ovary in these cases may not be normal. The acquired type may result from a uretosacral cellulitis, or from certain pelvic adhesions. The uterine angle is reduced below a right angle and a consequent abrupt bend in the curve of the uterocervical canal. The diagnosis is made by the finger on bimanual examination or by use of the uterine sound. All such cases are not necessarily pathological. The two main symptoms of pathological anteflexion are spasmodic dysmenorrhea and sterility. In the congenital variety the deficient development of the organ may account for the symptoms and not the acuteness of the angle. The treatment of the congenital type is at times discouraging. In a few cases pregnancy ensues and the condition is cured. Dilatation of the cervix usually gives but temporary relief. Electrical stimulation has proved discouraging. Stem pessaries help temporarily. In the chronic type of forward displacement the treatment is that adopted for pelvic inflammation.

Backward Displacement: The uterus may be displaced considerably backwards without in anyway interfering with its normal function. Every case of backward displacement is not pathological and does not require treatment. The condition is often incidentally discovered. The backward displaced uterus may or may not interfere with insemination. Many women with backward displaced uteri conceive and go through a normal pregnancy. The backward displaced uterus may encounter some difficulty in properly emptying itself during a menstrual period. A uterus with good muscular tone may empty itself without difficulties against gravity. In certain cases muscular spasms result with pain or dysmenorrhea. The retroverted uterus may cause varicose veins in the pampiniform plexus of the broad ligament. This is due to the broad ligament being folded backwards over the edges of the uterosacral ligaments. This causes stasis and engorgement of those veins. Varicose veins of the broad ligament can cause a dragging pain in the lower lumbar region. This becomes more marked premenstrually and menstrually due to the increase in the pelvic blood supply at that time.

Retroversion has often been found in the newborn baby. This congenital type may remain retroverted or may later in life be found corrected. At times one finds a patient with a retroversion today and in a few days' time the uterus is found in quite good position. Such uteri as these seem to have a marked degree of mobility and they doubtless change their positions many times during the patient's life, without causing symptoms.

The first degree of backward displacement is that in which the uterus is lying much in the same position as it is when being pushed backwards by a full bladder. This is spoken of as a retroposed uterus. The further degrees of retroversion will vary from that of a retroposed uterus to the uterus which is forward lying on the floor of the pouch of Douglas. We may therefore for clinical purposes consider

retroversion in three stages or degrees. The first stage being the retroposed uterus, the third stage being the extreme degree when the uterus is lying on the floor of the pouch of Douglas. The second stage refers to the position of the uterus when it is lying midway between these two positions.

The causes of acquired backward displacement may be grouped as follows: (a) relaxation of the uterine supports, (b) abnormal size and weight of uterus, (c) abnormal intra-abdominal pressure, (d) pelvic adhesions. The relaxation of the uterine supports along with an increase in size and weight of the uterus are the two factors which occur together post partum. It is therefore obvious why backward displacement is rather frequently found during this period of the maternity patient's convalescence. Backward displacements occurring at that time are usually amenable to simple treatment if such is instituted at the proper time. Doubtless many of these acute puerperal displacements do return to normal, yet many of them become chronic backward displacements and later give rise to symptoms.

Many backward displacements remain as simple backward displacements and symptomless. Others are complicated and are accompanied with symptoms. The most common complication is descent. This means that the displacement is accompanied with a certain degree of descent of the whole organ. Another complication is prolapse of the ovaries. Often one or both ovaries will be found lying on the floor of the pouch of Douglas. Pelvic inflammation is usually an associated condition and not a complication. This condition requires operative treatment for correction of the retroversion. A uterine fibroid is also an associated condition rather than a definite complication of the displacement.

The symptoms of backward displacement vary considerably. As stated above, many backward displacements do not cause symptoms. The usual symptoms are (a) pain in the sacral region and the lower abdomen, (b) menorrhagia, (c) dysmenorrhea, (d) leucorrhea, (e) sterility, (f) dyspareunia. These same symptoms are often found from other causes than backward displacements. The pain with backward displacement is not acute. It is usually in the form of a backache or a dragging sensation in the sacral region and in the lower abdomen. The pain is more marked when the ovaries are prolapsed. The menorrhagia may be the result of the accompanying endometritis. Dysmenorrhea is the result of the congestion along with the abnormal direction of the uterine canal. Sterility may only occur in a few cases of backward displacement. Urinary incontinence and bladder irritability if present are *not* due to the backward displacement.

The treatment of backward displacement of the uterus will vary somewhat according to the individual case. The treatment may however be conducted somewhat as follows: (1) In young unmarried women and in women who are long past the climacteric, if the case is uncomplicated, treatment is not required. (2) In sterile married women the displacement may be corrected even if no pelvic symptoms are present. (3) Cases occurring in married women in whom the ovaries are prolapsed usually require treatment. (4) Puerperal retroversions should always be corrected.

The actual treatment is essentially one of two forms: (a) pessary treatment, (b) operative treatment. Pessary treatment gives the best results in puerperal retroversions. In practically all other cases the pessary gives only temporary relief. The prolonged use of a pessary is objectionable, since it is difficult to keep clean and it may also cause ulceration. The pessary seldom ever cures a retrodisplacement except the puerperal cases. An operation suitably planned involves but little surgical risk and almost invariably presents a recurrence. A pessary used for puerperal retroversion should be inserted

reasonably early. It gives best results if used not later than the sixth or eighth week. A Smith or Hodge pessary is the one usually used. The patient takes a daily douche of a quart of warm water containing a teaspoonful of sodium bicarbonate. The pessary should be removed, cleaned and reinserted. At the end of about three of four months the pessary is removed and the patient goes one month without it. She then reports again for an examination. If the uterus is again out of position, the pessary may be tried for another month. At the end of this time it should remain in proper position without the pessary; if not, an operation is advised. Before attempting to replace a retrodisplaced uterus one should make sure that the lower bowel has been thoroughly emptied; also the bladder. In certain cases one may find it necessary to anesthetize the patient. Generally speaking this is not at all necessary.

The operation of choice for backward displacement is intraperitoneal shortening of the round ligaments, known as Gilliam's operation. This operation leaves the most normal uterovesical pouch. It also has the distinct advantage of not leaving any intra-abdominal structure around which the bowel may become entangled. One other distinct advantage is that if a pregnancy should follow it allows of practically normal and free mobility of the uterus upwards during its development. The uterus should *never* be fastened to the abdominal wall during the child-bearing period.

Prolapse is a complex condition which may involve several pelvic structures. There are five clinical varieties of prolapse: (a) Prolapse of the anterior vaginal wall (cystocele); (b) prolapse posterior vaginal wall; (c) prolapse of the anterior and posterior vaginal wall (cystocele and rectocele); (d) prolapse of the anterior vaginal wall and uterus; (e) complete prolapse of the uterus with inversion of the vagina (procidentia). Hypertrophic elongation of the cervix is a different condition and will not be considered here.

It is therefore clear that prolapse is not a displacement of the uterus alone, but usually the vaginal walls are involved in the descent as well as the uterus. In advanced cases we find that the bladder, rectum, fallopian tubes and ovaries, the broad ligaments, and even coils of intestine and omentum may form part of the prolapse.

Prolapse is really a hernia. It consists of a protrusion of the pelvic and abdominal contents through an aperture in the supporting structures. The coverings of the hernial sac are the vaginal walls and peritoneum. The hernial aperture is formed internally by the interval between the levator ani muscles and externally by the ostrum vaginae.

Prolapse is much more common in multiparous patients than in nulliparous patients. Mechanical deliveries is common history. Once the condition has begun it may be aggravated by increased intra-abdominal pressure, e.g. tumors, chronic cough and laborious occupations. Constipation and straining will also aggravate the condition. Prolapse of the uterus may be considered to occur in three degrees. The first degree involves descent of the uterus to a lower level than normal; but the cervix does not protrude at the vulva; it may or may not be accompanied with retrodisplacement. The second degree involves descent of the uterus still further until the cervix protrudes at the vulva and it is always accomplished with retrodisplacement of the uterus. In the third degree or procidentia, the whole uterus is expelled through the vulva, and the vagina is more or less completely inverted.

In long standing cases of second and third degree prolapse, certain secondary changes occur. The anterior vaginal wall loses its transverse folds and becomes smooth and looks more like a skin surface. The vaginal walls become thickened, hard and rigid. Ulceration is not uncommon in long standing cases, due to defective circulation, chronic irritation and friction. Infection may spread deeply, causing a

local peritonitis and the promotion of adhesions.

The condition of the perineal body is of importance in connection with prolapse. Three degrees of perineal laceration are usually described. The first degree involves half of the perineal body. The second degree extends up to, but does not include the sphincter ani muscle. In the third degree the sphincter ani has been severed, the anus is wholly or partly incompetent, and a portion of the rectal mucosa often protrudes. In rare cases perineal damage may not be found, but usually there is some degree of perineal. A partial or complete loss of tone of the perineal structures will predispose prolapse and incompetence is a distressing complication of prolapse.

Cystocele means a protrusion of the anterior vaginal wall, along with the base of the bladder and the urethra. The vaginal wall becomes stretched and smooth. The protrusion begins at the lowest part of the anterior vaginal wall and gradually comes to involve the upper or posterior section, finally pulling downwards on the cervix. The hernia of the bladder has occurred between the two pubococcygei muscles. Overstretching or tearing of these muscle fibers predisposes to cystocele. Commonly these patients have bladder friction disturbance, e.g. dribbling of urine when they laugh, cough or sneeze.

Rectocele is a protrusion of the lower part of the posterior vaginal wall which has carried with it a corresponding portion of the anterior rectal wall. This is the rarest of all the forms of prolapse. In procidentia the posterior vaginal wall usually separates itself from the rectal wall, so that the rectum does not actually take part in the prolapse. The loose arrangement of the cellular tissue between the vaginal and rectal walls allows of this stripping and separation of the two walls.

The symptoms of prolapse will necessarily depend upon the parts involved. Commonly there is a feeling of something coming down with or without a dragging pain. This is aggravated by standing or walking. At times in procidentia patients, their chief complaint is that of a protruding mass which is an inconvenience. Pain is most common during the early stages. The menstrual function and reproduction are usually not impaired except in cases of procidentia. Leucorrhea may or may not be present. When a cystocele is present there is usually some bladder instability and frequency of micturition. The patient often has dribbling when she laughs, coughs or sneezes. A cystitis may be present and in some cases a hydronephrosis is found due to impairment in the normal drainage of the ureter. Partial loss of control of the sphincter ani is not uncommon. One of the most common clinical symptoms is that the patient is incapacitated from leading a normal life.

Treatment of Prolapse: There are three lines of treatment to be followed: (1) rest and general massage; (2) pessaries; (3) operative treatment.

Rest and general massage is applicable to young patients in the post-partum period. With proper rest in bed and massage treatments, and helpful postures one may expect quite good results at the end of three or four weeks' treatment. This method is not of any use in long standing cases.

Long standing prolapse cases can never be cured with pessaries. The pessary is used only to relieve symptoms and to check the natural tendency of the condition to increase. The pessary is used mostly in the first and second degree cases. The watch spring pessary is the one commonly used. This can only be used when there is sufficient retentive power in the vaginal walls to retain the pessary in position. One usually selects the largest size pessary which can be worn with comfort. If the pessary is too large it will serve only to further stretch the vaginal walls and reduce their tone, thus aggravating the condition. The Napier pessary is at times resorted to in cases of procidentia when there is some good reason for not operating.

Operative Measures: The following give the most satisfactory results. (1) vaginal hysterectomy, (2) perineorrhaphy: bladder fixation and amputation of the cervix. (3) Le Fort's operation. Removal of the uterus alone in those cases is usually useless. The main part whichever procedure is carried out is to make certain that the pelvic diaphragm has been properly repaired.

Chronic inversion of the uterus is a very rare condition but needs at least mention under the heading of displacements of the uterus. In cases it is the result of an acute puerperal inversion. In a rarer number of cases it is found in association with a tumor, commonly a submucous fibroid. The puerperal cases are usually more complete than those associated with tumors.

The mucous surface is much altered, the openings of the fallopian tubes are obscured. Ulceration is common. In long standing cases the surface presenting is covered with squamous epithelium instead of columnar epithelium. The condition is usually accompanied with irregular hemorrhage. The differential diagnosis is made from (a) a large fibroid polypus, (2) carcinoma of cervix.

The treatment of chronic inversion of the uterus is carried out usually in one of two ways (a) repositor method, (b) operative method. The Aveling repositor may be tried in the beginning. This may be given a fair trial over a period of a few weeks, the position of the repositor being altered as required.

The operative measure of choice is vaginal hysterectomy in most cases of chronic inversion. If one is anxious to retain the uterus, the old method of incising the cuff and stitching up again after correcting the incision is well worth a trial; and it often gives quite good results.

PYELITIS, URETERITIS AND CYSTITIS CYSTICA*

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MORGAGNI in 1761 was the first to describe cysts of the mucous membrane of the renal pelvis and ureter. He reported 2 cases, each showing a hydroureter and hydronephrosis, bilateral. Johnson, in 1816 reported a third case. Rayer, in 1837 and Rokitansky in 1861 reported cases and stated that cysts appear to be more frequent in the urinary passages than they are in or upon other excretory ducts. Virchow in 1863 compared the cysts to the mucous cysts of the vagina.

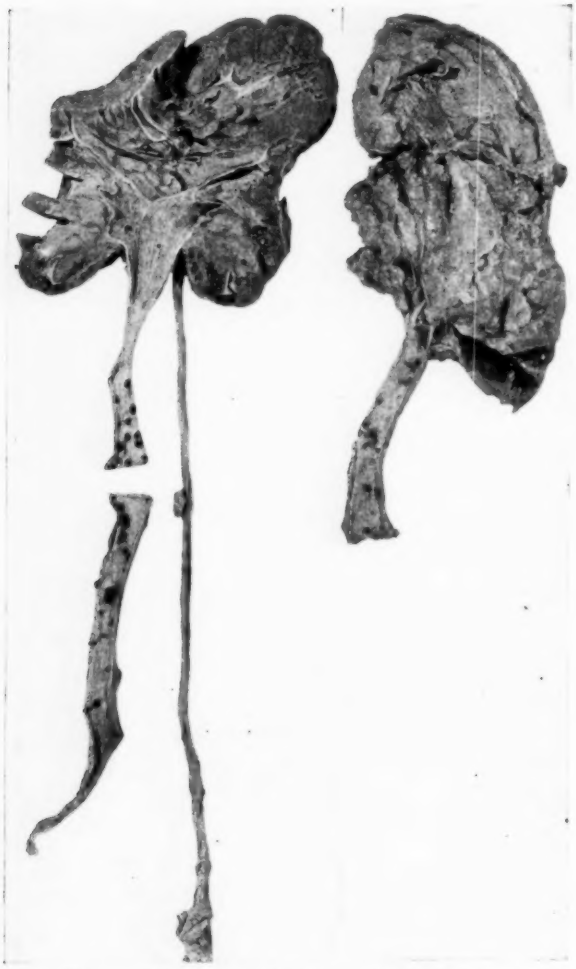

FIG. 1. Kidneys and ureters showing cysts.

Litten in 1876 first studied these cysts microscopically and concluded that they were formed by an inflammatory blocking of the mucous glands and crypts of the mucous membrane, the retention of the secretion in these glands and crypts forming the cysts.

In 1889, Eve was convinced that they were parasitic in origin. He was supported by Morris, Bland-Sutton, Clark, Voelcker and Pisenti.

Von Brunn in 1893 described epithelial bodies (buds or sprouts) lying below the mucous membrane with an epithelial connection with the surface epithelium, and epithelial bodies showing no connection with the surface epithelium, which he designated as epithelial cell nests. These epithelial cell nests and buds are formed by the downward proliferation of the epithelium in the form of sprouts and their subsequent separation from the mucosal lining. He believed that this proliferation of epithelium was stimulated by inflammation.

Lubarsch in 1893 and Aschoff in 1894 confirmed Von Brunn's observations. Marchwald found ureteritis and cystitis cystica in a newborn, and maintained that an inflammatory process was not necessary to the formation of Von Brunn's epithelial buds or cell nests.

Stoerk, after a careful study of 13 cases of ureteritis and cystitis cystica, differed with Marchwald in that he considered the origin of the Von Brunn's cell nests to be inflammatory, and considered the cystic formation to be a secondary phenomenon.

Giani produced cystitis cystica experimentally in rabbits by the irritation of a foreign body in the bladder, and by curretting the vesical mucosa through a suprapubic cystotomy.

Morse in 1927 reviewed the literature and

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Read before the North Central Branch, American Urological Association, Rochester, Minn., November 22, 1929.

reported 3 cases from the Mayo Clinic with a careful microscopical study of the kidneys, ureters and bladder in 125 necropsies.

Fig. 2. Microphotograph of right ureter, showing cyst lined by cuboidal and columnar epithelial cells.

He found the so-called buds or cell nests of Von Brunn in 108 of the 125 cases or 86.4 per cent. In 63 of the 108 cases in which the epithelial buds or cell nests were present, inflammation was manifested by lymphatic infiltration or fibrous tissue proliferation.

Joelson in 1929 reported a case clinically diagnosed, associated with bilateral nephrolithiasis.

CASE REPORT

E. V. White, female, age fifty-eight years, Baltimore City Hospital, Autopsy No. 2604.

Clinical Diagnosis: chronic pyonephrosis and cystitis.

Urinary Tract: the bladder wall measuring 10 to 14 mm. in thickness with the mucosa being of a dark red color with 3 small yellowish brown cysts measuring 3 mm. in diameter just above the trigon. The left ureter was dilated, measuring 10 to 15 mm. in diameter. On section the wall measures 3 mm. in thickness, mucosa smooth and glistening with a number of bluish brown cysts measuring 1 to 3 mm. in diameter on its surface. There is a complete duplication of the right kidney pelvis and ureter, the ureter, draining the lower pelvis, is dilated, measuring 10 to 18 mm. in diameter. On section the wall is thickened, measuring 2 to 4 mm. with the mucosa dark red in color with a number of brown cysts on its surface, measuring 2 to 6 mm. in diameter. The upper right ureter on section shows no gross changes.

Kidneys: the capsule strips off with increased resistance, leaving a dark red granular surface. On section the cortex and medullary portion are thinned out with the architecture of the kidney poorly preserved.

The pelvis and calices are dilated and filled

with purulent urine, with the mucosa in the lower right pelvis dark red in color. Small brown cysts measuring 2 to 4 mm. in diameter are seen on the mucosa of the left kidney pelvis and lower right kidney pelvis.

Microscopical Examination. *Kidneys:* sections show a thickening of the capsule due to adult connective tissue cells; just beneath the capsule in the cortical zone, collections of small round cells and polymorphonuclear leucocytes are seen. The capsule of Bowman shows a definite thickening due to adult fibrous connective tissue cells, some of which are stained as a pale pink homogeneous mass. The glomerular tufts in areas are stained as a pale pink homogeneous mass; in other areas they show the epithelial and endothelial cells poorly stained with the capillaries filled with leucocytes and red blood cells. The space between the capsule of Bowman and the glomerular tufts is filled with coagulated serum and fragmented cells. A number of tufts are replaced by fibrous connective tissue. In areas the glomerular tufts are seen to show their epithelial and endothelial cells well preserved. The tubules in many areas show the tubular epithelial cells poorly stained with fragmented cells and leucocytes free in the lumen. In areas, the tubular epithelial cells are distinct in cell outline with a well-stained nucleus. Between the tubules, the arterioles show moderate thickening of the tunica intima with a perivascular infiltration of small round cells, polymorphonuclear leucocytes and plasma cells. Sections from the pelvis show the surface epithelium to be swollen and finely granular in appearance. Just beneath the mucosa, a cyst is seen, lined with one or two layers of cuboidal epithelial cells well stained, with the lumen filled with coagulated sera, fragmented cells and a few polymorphonuclear leucocytes. In the tunica propria and muscular layer, an infiltration of small round cells are seen with an occasional plasma cell.

Ureter: the muscular layer shows the circular fibers and longitudinal fibers well stained with a perivascular infiltration of lymphocytes. The tunica propria shows the elastic fibers poorly stained, with collections of small round cells between the elastic fibers. In several sections, collections of epithelial cells are seen cuboidal to columnar in type, surrounded by fibrous connective tissue cells, in areas cysts are seen lined with one or two layers of cuboidal epithelial cells with the lumen filled with coagulated sera, fragmented epithelial cells and a few polymorphonuclear leucocytes. The cysts are just beneath the surface epithelium which are well preserved. In several sections epithelial cell nests are seen communicating with the surface epithelium.

Bladder: Sections from the trigone show marked thickening of the muscular layer, due to fibrous connective tissue proliferation between the muscle fibers. The tunica propria shows marked infiltration of lymphoid cells and polymorphonuclear leucocytes. One cyst is seen which is lined by cuboidal epithelial cells, the lumen filled with fragmented cells and coagulated serum. The mucosa shows the transitional epithelial cells swollen and finely granular in appearance with necrotic material on its surface.

DISCUSSION

From the review of the literature and a study of this case, the author concludes that cystitis, ureteritis and pyelitis cystica are inflammatory in origin as described by Von Brunn in 1893. [For bibliography see author's reprints.]

INFLUENZAL FASCITIS

WITH A REPORT OF FIVE CASES*

SAMUEL KLEINBERG, M.D.

NEW YORK

SEVERAL years ago I observed a few cases in which a painful swelling appeared in the feet and heel cords following an attack of the grippe. During each succeeding winter I came across one or more similar cases and I finally became impressed with the fact that I was dealing with a definite complication of the grippe or influenza. This sequel of influenza undoubtedly occurs frequently, but has not attracted attention probably because its symptoms are usually mild, cause only moderate and temporary disability and no permanent defect. I am prompted to report my cases because I have found no reference to this lesion in any of the standard textbook articles on influenza. The following group of cases came under my observation during last spring and this year. All of them occurred during the months when the grippe was prevalent.

CASE REPORTS

CASE I. Anna W. (Hospital No. A23-076), twenty-eight years old, was admitted to the service of Dr. Royal Whitman on May 6, 1927. Her chief complaint was pain in her feet. Three weeks previously she was ill with the grippe. She had a moderate rise of temperature and was confined to bed for two days. She was gradually recovering from the general depressing effects of the grippe when her feet became painful. Three days before her admission to the hospital the pain was very severe, and on the following day a swelling appeared in each foot making walking uncomfortable and difficult.

Upon her admission to the hospital she was unable to walk. She had a marked pallor, such as is common following the grippe. In the right foot there were two swollen areas, one on the inner and the other on the outer side of the ankle. The inner swelling was about 5 by 3 in. in area. It involved the astragalonavicular region and extended upward to above the malleolus. The swelling was not well defined but faded gradually into normal tissue. The skin in the affected part was bright red, hot, glossy and very sensitive to pressure. Upon palpation it was evident that the subcutaneous tissues were swollen. There was a similar but less marked lesion on the outer side below the malleolus. The swelling did not extend to the sole of the foot but the plantar tissues were sensitive to pressure.

In the left foot there was a red, swollen, painful area on the inner side of the ankle, chiefly in the region of the attachment of the tibialis anticus tendon, and also a small painful swelling on the outer side of the ankle along the peroneus brevis.

The patient was kept in bed and given large doses of tolysin for her pain. Large quantities of fluids were given by mouth to counteract the general depletion. The temperature fluctuated between 100 and 102.8°F. Within five days her fever subsided and the local symptoms disappeared. On the eleventh day she was discharged cured. Two blood cultures were negative, the blood chemistry was normal, the urine showed no abnormal findings and the blood count was negative except for a moderate anemia.

This patient had, I believe, an acute inflammation of the subcutaneous fascial tissues, a fascitis, on the lateral surfaces of both feet and in the plantar region of the right foot, secondary to an influenza and probably due to the same bacterium.

CASE II. Edward G. twenty-eight years old was treated at the Hospital for Ruptured and Crippled (Case No. B1-919) on the service of Dr. Royal Whitman. He was admitted on April 19, 1927 complaining of pain in the back of the left knee, the soles of both feet and the right hand. These symptoms came on after an illness that probably was the grippe, but may have been a tonsilitis.

Examination upon admission to the hospital showed that the patient was in good general condition. There was a painful swelling in the plantar region of each foot. The skin was not red

*Read before the Section on Orthopedic Surgery, New York Academy of Medicine, April 19, 1929.

or hot, but the swelling was very sensitive to pressure. The skin was glossy and evidently stretched by the swollen subcutaneous tissues.

Examination at the hospital showed that only the feet were affected. There was a swelling in the middle of the plantar region of the

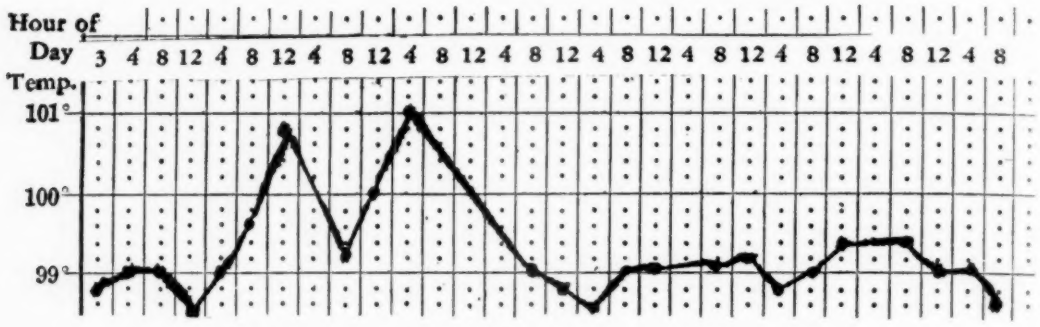

Fig. 1. Usual temperature curve in typical case of influenzal fascitis.

The swelling was not well defined and faded gradually into the surrounding area. There was also sensitiveness to pressure on the tissues along the tendons of the internal hamstring muscles of the left knee, although there was no visible or palpable swelling in this region. The interphalangeal joints of the fingers were swollen and painful, but without any appreciable restriction of motion. One gained the distinct impression that the swelling in the fingers was periarthritic.

The patient was kept in bed, given tolysin, large quantities of fluids by mouth, colonic irrigations and baking and massage locally. In four days all his symptoms disappeared and he was discharged. Subsequently he had a temporary relapse which was readily controlled by rest.

This patient had apparently a subacute inflammation of the fascial tissues in the soles of both feet, about the left internal hamstrings and about the finger joints, that is, multiple foci of fascitis probably due to the organism causing influenza.

Case III. William S. (Hospital No. A23-076), thirty-three years old, was admitted to the service of Dr. Royal Whitman at the New York Hospital for Ruptured and Crippled on April 29, 1927. He complained of painful swellings in the soles of his feet and about the heel cords. He was well until a week before his admission to the hospital, when he had an attack of the grippe. After an illness of a few days and when he was apparently recovering from the grippe his feet became painful. On the day prior to his admission to the hospital his temperature was 101°F. and he had to walk on the ball of his toes because of pain in his feet. right foot. The skin over the swollen tissues was not red, but was warm. Pressure over the swelling caused intense pain. The subcutaneous tissues about the lower third of the heel cord were also markedly swollen. The skin was normal in color, but decidedly warm and sensitive to pressure. There were similar lesions in the left foot.

This patient had an inflammation of the subcutaneous fascial structures in the plantar regions of the feet and about the heel cords. He was kept in bed, given tolysin, colonic irrigations and large quantities of fluids to counteract the toxemia and depressing effects of the grippe. His fever subsided gradually and he was discharged cured at the end of a week.

Case IV. (Hospital for Joint Diseases, No. 19617R.) This is the severest case in my series. Mrs. H. came under my care on January 10, 1929. Two weeks previously she had a mild cold or grippe. A week later she had a severe chill, a marked rise in temperature and intense pain in the lower part of her back. Several days later the backache subsided, but a very painful red swelling appeared on the inner side of the right foot and ankle. She also had severe pain in the upper part of the right thigh in the region of the great trochanter, and in the left side of the neck. She had a daily rise of temperature to about 103°F. The pain was so marked that she got very little sleep. Even morphine hypodermatically did not give her much relief.

At my first visit I found her very restless, tossing about trying to find a comfortable position for her legs. She was very pale and apparently exhausted from loss of sleep. There was a large red, hot, painful swelling on the inner side of the right foot. This extended

from the inner border to well above the malleolus. It occupied the entire inner aspect of the ankle. The swelling was diffuse and faded gradually into surrounding normal tissue. On the outer aspect of the ankle the swelling was similar, but less extensive. The skin was bright red and glossy. There was also a moderate swelling about the lower part of the heel cord. The skin here was not red. The ankle motions were restricted by the pain in the swollen areas, but the ankle joint itself was not involved. The swollen areas were very sensitive even to light pressure. The tissues over the left sternomastoid muscle, the right great trochanter and the lumbosacral joint were tender to touch. There was no discernible swelling or redness of the skin in these latter areas. Tolysin, aspirin and even morphine gave little relief.

On the following day a red, hot, very painful swelling was found on the inner side of the left foot similar in appearance and distribution to that on the right foot. The tissues about the left heel cord were also swollen, sensitive to pressure and painful during motions of the foot, especially dorsiflexion. A painful spot appeared on the outer side of the left thigh slightly below and behind the great trochanter. Application of heat externally with a baking lamp increased the pain. As the symptoms were becoming worse and the pain was intense, it was determined to remove her to a hospital for observation, and she was admitted to my service at the Hospital for Joint Diseases. *X*-ray pictures of both feet and a blood count were negative. It occurred to me to apply wet dressings of a boric acid solution to the swollen parts. These were changed every hour, day and night and were kept continually covered with oiled silk. This procedure worked like a charm. The pain immediately subsided. The swelling, redness and sensitiveness rapidly diminished. A slight fever persisted for about a week. On the third day after admission to the hospital the tissues about the sternomastoid and the attachment of the left adductor muscles to the pelvis became painful and sensitive to touch. There was no redness or swelling in these areas. These symptoms lasted only several days. The urine was negative except for a trace of albumin. A blood culture was negative after seventy-two hours of incubation.

The patient was discharged on the eighth day after her admission, because she had to attend the funeral of her mother. When she left the hospital there were no symptoms in either lower limb, but her temperature was 99.5°F., indicating that she was not entirely well. Two days later she returned on account of a slight relapse of the swelling, redness and pain in her feet. The application of wet boric acid dressings gave immediate relief. In five days her temperature was normal and all symptoms disappeared. She left the hospital and has remained well since.

Case v. Mr. B. came to my office complaining of a painful swelling on the front of his left leg. This extended upward from the ankle for about 4 in. The symptoms which came on about a week after a mild cold or grippe during the recent epidemic of influenza in New York, had existed for about five days when he consulted me, and they were getting worse.

Upon examination I found a diffuse swelling on the front of the lower part of the leg. There was no redness, but there was slight local heat and marked sensitiveness to pressure. An *x*-ray picture was negative. He was immediately urged to rest in bed and apply boric acid dressings. He did so and obtained complete relief and a cure in about six days.

The cases described have many common characteristics. All of them were adults and were ill when the grippe was prevalent in the city. In all the patients, including those observed prior to 1927 and not reported because I had not kept a record of them, the symptoms came on from four to seven days after an attack of the grippe. The grippe had not been of a severe nature in any of the patients.

The local lesion occurs most often in the feet, but may occur in the subcutaneous fascial tissues of any part of the body. The characteristic clinical appearance is a painful swelling in the sole of the foot and around the heel cord, that is, in the plantar fascia and the areolar tissues about the tendo Achillis. Elsewhere it occurs in the neighborhood of tendons and subtendinous bursae. When the lesion is in the lower limb walking is difficult or impossible, and the pain is increased by any movement of the limb. Pressure upon the swollen area causes pain. The skin is usually white and shiny. When the inflammation involves the skin, it is red, hot,

glossy and very sensitive to pressure. The essential pathologic change is, I believe, an inflammation of the subcutaneous fascial tissues. Since the lesion has been found only in patients who have or are just getting over an attack of the grippe or influenza, it appears to me logical to at least assume that it is due to an invasion of the fascial structure by the bacterium of influenza or its toxins. I have had no opportunity to obtain bacteriological proof of this conclusion which I, consequently, base on clinical evidence only. As the inflammation of the subcutaneous tissues is a complication of influenza, I believe that the term influenzal fascitis is appropriate since it describes the pathology of the lesion and its likely etiology.

The treatment, from my experience in the last two cases, is fortunately simple. It consists of rest in bed and the application of wet dressings to the inflamed parts. The inflammation subsides in a few days and leaves no residual defects. From the relapse of the painful swelling in some of the cases it is advisable to continue rest in bed for at least a week after freedom from all symptoms.

DISCUSSION

DR. A. DEF. SMITH: I would like to know why Dr. Kleinberg is so certain that this inflammation is confined to the fascia and subcutaneous tissues. There are other tissues in the same regions, tendon sheaths, muscles, joints and periosteum, and I don't see why they may not be involved as well as the fascia. There is not any pathological proof, of course, that it is the fascia.

DR. RALPH C. KAHLE: I have had the good fortune of seeing several of these cases of Dr. Kleinberg's and have had the opportunity of observing a case of my own, a man thirty-six years old, with a typical history of four days after the initial grippe developing symptoms with swelling of the inner side of the ankle which subsided after rest and applications of warm boric acid solution.

This afternoon at the Cornell Clinic I saw another case with a similar history, six days after the grippe, with a swelling on the inner side of the ankle. It was just as Dr. Kleinberg described, hard, tender, reddened, etc. There was nothing present to make any one think the lymphatics were involved nor were the signs of venous thrombosis. For want of another term, I called it the same as Dr. Kleinberg and used the same treatment as in the other case.

DR. CHARLTON WALLACE: How can one tell the difference between fascitis and cellulitis?

DR. SIGMUND EPSTEIN: The history of grippe is very often erroneous. A case of painful foot and calf was seen yesterday; three doctors said that he had grippe: a severe phlebitis of the calf branches was the real diagnosis. His troubles had lasted five weeks. I find similar swellings of the foot, ankle and tendo Achilles are very common. Some of them are red. They consist of subcutaneous indurations and are infectious in origin. The British call them fibrositis; the dermatologists call them erythema nodosum.

DR. KLEINBERG, closing: I am sure that the muscles in cases of influenzal fascitis are not involved. In most of the cases the swelling is limited to the sole of the foot and there is no pain on contraction of any of the muscles of the foot or leg except when the motion causes tension on the swollen area. In several cases the swelling extended over the external malleolus and it is often in the region of the heel cord where there are no muscles. When one considers the composite picture of influenzal fascitis, one readily concludes that there is no involvement of the muscles. The inflammation is usually limited to the subcutaneous tissues; occasionally there is involvement of the skin. So far as cellulitis is concerned there is no difficulty in differentiating fascitis from the ordinary type of cellulitis. In the latter condition, there is an enlargement of the glands, a lymphangitis and a severe toxemia. These are absent in fascitis. Periostitis can be excluded, first, by the fact that the *x*-ray pictures are negative and secondly, by the fact that pain upon pressure is present even to light touch when manifestly there is no irritation of the periosteum. I have not seen any untoward sequelae following influenzal fascitis.

GLUCOSE IN THE TREATMENT OF TOXEMIAS OF PREGNANCY*

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A LIST of the various theories of the etiology of the toxemias of pregnancy would consume more space than is allotted for this entire paper. An enumeration of the many procedures employed in the treatment of these conditions would be equally voluminous. We will, therefore, limit our discussion to a consideration of carbohydrate metabolism in these toxic conditions, and some observations on the phenomena encountered in the administration of glucose (or dextrose) in the treatment thereof.

The most common forms of toxemia of pregnancy are the vomiting of the early months of gestation, culminating in hyperemesis gravidarum, and the disturbances of the latter months, terminating in eclampsia.

The contributing factors in the etiology of the early toxemias are many.

The immediate cause of such disturbance is the presence in the blood stream of substances acting as toxic agents exerting a deleterious influence upon metabolism, with the resultant digestive disorders.

In the average case, the reserve forces are sufficient to adjust these difficulties in a few weeks, so that the patient will continue her pregnancy with a good degree of comfort and well-being.

In some few cases the distributing element is of such a severe nature that the maternal organism is not able to successfully combat these evil forces, the nausea and vomiting become more and more severe, until nothing is retained in the stomach.

This extreme condition, fortunately, is not so common as formerly, due largely to better supervision of the patient and more energetic efforts to correct the condition producing the ordinary vomiting of pregnancy. Few cases indeed need progress to the state of hyperemesis gravidarum, if proper attention is paid to the earlier and milder forms of vomiting.

It has been shown by Paul Titus[1] and others that in practically all of these cases, whether mild or severe, a marked carbohydrate disturbance is present. As soon as pregnancy is established, the carbohydrate needs of the mother are markedly increased. When the endocrines governing metabolism are able to care for this sudden need, there is no marked disturbance; when, however, the requirements develop more rapidly than the metabolic hormones are able to adjust themselves to the new conditions, digestive disturbances follow.

The developing placenta requires a great amount of glycogen,[2] and the liver is called upon for an increased supply. In multiple pregnancy the placental area and volume being greater, a still larger demand is made upon the store of glycogen and a greater tax is put upon the endocrines. If the metabolic forces are not able to supply the liver with sufficient sugar to insure an adequate glycogen reserve, hepatic degeneration results, and glycogen production falls, even below its ordinary level. Thus a vicious circle is thereby established. The more the liver is damaged, the less glycogen is produced, and the more the glycogen reserve is depleted, the more liver damage ensues.[3]

It is probably this sudden need that first throws the endocrines out of balance, and once disorganized, they in turn fall below ordinary efficiency.[4]

Before this phenomenon was so well understood, the usual method of treatment of hyperemesis gravidarum was an early

*Read before The American Association of Obstetricians, Gynecologists and Abdominal Surgeons, Memphis, Tenn., September, 1929.

emptying of the uterus. This method of necessity sacrificed the fetus and frequently the mother, since by the time the disturbance progressed to the stage of actual hyperemesis, the maternal condition was such that the shock of abortion was sufficient to produce a fatal issue for the mother. The problem in the treatment of this condition, therefore, was to supply glucose to the maternal organism in such a form and in sufficient quantities, that the liver would have a chance to recuperate, the endocrines have opportunity to adjust their functions to the new requirements, the maternal organism would be adequately nourished, the dehydration combated, and the fetal needs met.

The fact that the employment of such a system of treatment for hyperemesis is successful, both regarding mother and child, making therapeutic abortion seldom necessary, seems to prove its efficiency.

While various modifications in the technic are recommended by different operators, yet the glucose treatment of hyperemesis has been developed to a point where the essential features form a common basis for these methods.[5]

The following case is typical of the condition under consideration and the treatment employed, and is here reported as an illustration of the usual results obtained by us in hyperemesis:

Case 1. Mrs. M. S. B., para II, aged thirty-seven, was referred to me on June 9, 1928. Examination showed patient to be dehydrated, dry, parched skin, heart rate 130, temperature 100, blood pressure 120–85, urine negative, for sugar, albumin, casts or pus. She gave a history of having her last menstrual period 3 months previously (March 5), of being able to retain practically nothing by stomach for the past week. Complained of severe epigastric pain, sleeplessness, diplopia, pronounced weakness and prostration.

Vaginal examination revealed a much larger uterus than would be found in a 3 months' pregnancy.

Treatment. One ampule of corpus leutea was immediately given intravenously, without any beneficial results.

She was removed to the Kentucky Baptist Hospital. Everything by mouth was discontinued, 1000 c.c. of 5 per cent glucose in 1 per cent. saline were given intravenously, the administration being completed in forty-five minutes. This was repeated daily for five days, when the urinary output reached the amount of in-take; 4 oz. of 10 per cent glucose containing 60 grains of potassium bromide were instilled into the rectum every four hours.

The nausea and vomiting stopped immediately after the first injection of glucose: the abdominal pain ceased with fifteen minutes; neither of these symptoms recurred.

On the third day some vaginal bleeding and cramps occurred, but subsided two days later. The diplopia disappeared about the seventh day. The pulse and temperature returned to normal on the third day.

From the third day fluids were given by mouth, at which time the rectal instillations were discontinued, and elixir bromidorum and elixir luminal, each drams 1, were given by mouth three times daily. After the fifth injection of glucose the temperature went up to 102 and the pulse to 120, remaining thus for several hours, then returning to normal. Soft diet was given after the fifth day, and regular diet on the seventh.

The patient left the hospital on the tenth day in good condition, with no recurrence of symptoms.

As pregnancy progressed, hydramnios developed so markedly that no diagnosis of position or presentation was possible by auscultation or palpation. On September 5, twin pregnancy was diagnosed by x-ray, showing about seven months' pregnancy. She was readmitted to the hospital on October 10, because of discomfort due to abdominal enlargement.

By November 1, the hydramnios had become so marked that the respiratory and circulatory embarrassment demanded interruption of pregnancy.

The babies were both female, weighed 4 lbs. 6 oz., and 5 lbs. 6 oz., respectively, apparently single-ovum twins, well-developed and healthy. Mother and babies left the hospital in good condition, and today, more than six months later, are all thriving and well.

Another interesting case was one of hyperemesis in an unmarried primipara, who was also suffering from an acute nephritis due probably to the taking of

large amounts of turpentine with abortive intent.

Case II. This patient, R. C., para 1, fourteen years old, four months' pregnant, was brought into the Louisville City Hospital in a semi-comatose condition. Her history showed she had taken 1 oz. of turpentine several times in the three weeks preceding admission.

She was given ten administrations of glucose without relief of the emesis.

A therapeutic abortion was done twenty-two days after admission, after which vomiting ceased, and patient recovered.

The blood chemistry taken at intervals during this period showed: non-protein nitrogen, 75, 93.6, 194.8, 113, 85.5, 88.2; urea, 38, 70, 88.2, 70, 40, 46; creatinine, 8, 7.5, 10, 9, 6, 4.8; plasma bicarb., 73, 150, 140, 130. An initial blood-sugar reading showed 153.

It will be seen that the blood chemistry in this case showed improvement under glucose, even though the vomiting was not controlled thereby. We have seen other such cases showing severe vomiting, accompanied by acute nephritis, in which glucose did not relieve the vomiting. In all cases of hyperemesis, however, unaccompanied by an acute nephritic groundwork, glucose has been eminently successful.

The 2 cases above reported illustrate the results obtained by glucose in hyperemesis, first, when the hyperemesis has for its primary basis a carbohydrate deficiency due to metabolic upset, and, as in the latter instance, where the vomiting is incidental and the basic condition is an acute damage to the other organs, especially the kidneys.

Regarding the rôle that carbohydrate deficiency plays in the late toxemias of pregnancy, much valuable work has been done recently, and there is a strong probability that out of this will soon be developed more definite knowledge of the relationship of carbohydrate metabolism to eclamptic conditions.

Paul Titus[6] has brought out some very pertinent points in this connection, one of the most striking of these being his findings in blood-sugar levels during the acute eclamptic state. He has shown that the convulsions are immediately preceded by a sudden marked drop in blood-sugar; that the hypoglycemia in eclampsia is relative and not absolute: that is, that the average blood-sugar level in this condition is not necessarily lowered, but drops markedly only in relation to a convulsion. He further suggests:

> The chief difference between the clinical symptoms of the toxicoses of early and late pregnancy is due to the fact that the former is a slow, and the latter an acute, rapidly progressing process. In early pregnancy the fetal requirements are less and the demands are more gradual, so that the glycogen depletion of hyperemesis rarely is swift enough to cause convulsions. Occasionally, however, we see that fulminating process termed acute yellow atrophy of the liver which is pathologically identical with hyperemesis but is so acute that eclampsia-like convulsions usually occur.[7]

In our own experience there would seem to be some evidence of such a relationship as Titus describes. The chief clinical differences in the early and late toxicoses, as we have observed them, are the marked elevation of blood-pressure, albuminuria, and convulsions, present in eclampsia, while absent or negligible in hyperemesis. In eclampsia we have also failed to get from the administration of glucose the phenomenal benefits obtained in the treatment of hyperemesis. As to liver pathology, we have not found in all fatal cases of eclampsia the typical acute yellow atrophy lesions of this fulminating toxemia of early pregnancy.[8] In view of the above variations, the question arises if the differences encountered in early and late toxemias would not have to be accounted for by a more basic factor than the mere element of rapidity of development. For example, would the rate of the process account for the elevation of blood-pressure in eclampsia, while normal in hyperemesis? It would seem to the writer that the severe manifestations of hyperemesis are more nearly the result of glycogen depletion,

TABLE I

Name	Para.	Age	Diagnosis	Glucose given		Blood-pressure before glucose administration	Blood-pressure 1 hour later	Blood-sugar before glucose administration	Blood-sugar 1 hour later
				c.c.	Per cent				
L. P.	V	29	Eclampsia	500	10	205/120	200/110		
				500	10	185/120	185/110		
J. D.	VI	35	Eclampsia	450	10	185/120	175/100	97.5	166.
E. P.	I		Eclampsia	500	20	148/98	142/90		
H. M.	VI	38	Eclampsia	500	20	138/90	140/80		
				500	20	170/90	150/80		
M. P.	III	27	Eclampsia	500	20	230/110	210/106		
				500	20	150/100	140/100		
				400	20	199/106	196/103	111	200.
E. G.	I	17	Eclampsia	500	12	170/110	130/80		
K. W.	I	21	Eclampsia	500	20	152/105	150/105	111	112
A. J.	X	31	Eclampsia	250	20	180/140	140/105		
				200	20	184/124	174/120		
				500	20	190/125	170/115	80	88.3

The average show a drop in blood-pressure of 12 points in the systolic and 9 points in the distolic pressure. Blood-sugar shows a rise of 40.5 points.

while the carbohydrate disturbances of eclampsia are more directly the result of the toxemia, and occur as a part of the general metabolic upset; or in other words, an incidental occurrence in eclampsia rather than the primary manifestation as in the early toxemia.

We have recently been interested in the influence of intravenous glucose upon blood pressure and blood-sugar in eclampsia. In the accompanying table, it will be seen that both the systolic and diastolic pressure were lowered in practically every instance and no elevation in any case.

The average for all cases showed a lowering of 12 mm. for the systolic and 9 mm. for the diastolic. The blood-sugar, however, was elevated in each instance, the average being 40.5 points. In each of these cases, blood-pressure readings were taken at frequent intervals after the discontinuance of the glucose administration. Before the glucose was given the reading was 180/140; 15 minutes after the completion of administration it was 140/100; after 25 minutes, 160/100; after 35 minutes 155/110; after 45 minutes 150/100; one hour after discontinuance the reading was 140/105. This patient passed 1000 c.c. of urine within one and one-half hours after glucose administration. We have noticed in practically all eclamptic cases that the urine is more copious after glucose administration. This may account in part for the fact that the blood-pressure is lowered by the glucose, even though the blood volume is temporarily increased. Yet we believe it would not wholly account for this phenomenon. The blood-pressure is lowered before the kidneys would have time to react to this extent. There must be a neutralizing of the vaso-constrictor effect of the toxin, whatever that toxin may be.

The writer would hesitate to add even one more theory to the already long list of speculations regarding toxic action in eclampsia, but in an earnest effort to account for the various phenomena observed in the treatment of this condition, one naturally seeks to find some plausible explanation, and a number of observations already accepted as authentic would, to our mind, offer a possible reason for certain clinical results in the employment of glucose.

In the first place, blood-sugar falls rather suddenly immediately preceding a convulsion. Then the eclamptic convulsion is very similar to the manifestations seen in shock from an over-dose of insulin.[9] Also, blood-pressure is lowered by glucose administration, even though blood-volume is temporarily increased. The use of glucose, too, seems to have a marked influence in lessening the number of convulsions. Therefore, it would seem reasonable to conclude that the toxin of eclampsia stimulates the production of insulin, and that the convulsions are precipitated, in part at least, by the sudden appearance in the blood stream of an overload of insulin. If this be true, the beneficial influence of glucose could be accounted for in part by its neutralizing effect upon this insulin.

As to the amount of glucose used, it will be seen by the accompanying table, 500 c.c. of a 20 per cent solution were usually employed. In instances where we have used 1000 c.c. of a 10 per cent solution, results were not markedly different.

As to the amount of glucose which may be tolerated by toxic patients some very interesting work has been recently done by Dr. G. A. Hendon.[10] He uses a continuous flow of glucose solution which he styles "venoclysis." It has been the writer's privilege to observe a number of cases treated by this method, and the results obtained have been spectacular. This procedure is as yet in a more or less experimental stage, but enough work has already been done to demonstrate some very interesting facts regarding the intravenous administration of glucose.

To Dr. Rudolph Matas probably belongs the credit of first calling attention to this method. In June, 1923, he reads a paper before the American Surgical Section on "Continuous Intravenous Drip."[11] The method proposed by Matas did not come into popular use, however, probably due to the following difficulties: (1), inability to keep the solution at an even temperature; (2), inconvenience to the patient, the arm having to be held immobile during the administration; (3), the clogging of the needle or canula by the solutions.

These difficulties have apparently been obviated by the technic and apparatus employed by Hendon. He has used this form of administration in something over 100 cases. A 5 to 10 per cent solution of anhydrous glucose in freshly distilled water is the concentration usually employed. The average amount given an adult is 4000 c.c., or one gallon, every twenty-four hours. The average duration of the continuous administration in all his cases is about seven days, one patient, a gastric ulcer case, receiving 5000 c.c. of a 5 per cent daily for sixteen days.

In none of these patients has there been any untoward results; there have been no symptoms of hyperinsulinism (a probable complication suggested by Thalheimer),[12] except in two hyperemesis cases where insulin was given with the glucose. The two patients showed evidence of slight insulin poisoning, which disappeared immediately upon discontinuing the insulin, the glucose being continued.

The concentration of the solution employed has been determined by the point at which sugar appears in the urine. If glycosuria appeared, the concentration is reduced. Practically all of them tolerate a 5 per cent strength, and many of them 10 per cent without glycosuria. In those cases where blood-sugar determination has been done, the average level has been about 150, the highest reading being 169. In this case, however, no sugar showed in the urine.

One reason why such apparently large quantities can be taken is that no sudden load is put upon the powers of assimilation, or upon the circulatory function. The usual rate of continuous administration is one drop per heart beat.

Woodyatt, Sansum and Wilder[13] claim that a normal individual can assimilate without glycosuria 0.9 gm. of glucose

per k. of body weight per hour. In other words, a man weighing 150 lbs. would take care of 2.5 lbs. per day.

In Hendon's series the patients received from 1 to 2 lbs. per day.

The cases in which this method has been employed include those of severe toxemia, septicemia and conditions in which oral feeding has, for any reason, been impracticable. These patients require no water or food by mouth during the administration.

A number of these cases have been those of desperate hyperemesis gravidarum, in which the use of glucose by the usual methods was unsuccessful, while with the continuous drip the condition was completely relieved.

The essential features of the apparatus used are two thermos bottles of 1000 c.c. capacity each, suspended in a wire frame and attached to a Y-tube, which is in turn connected to a Murphy drip tube; from this a single rubber tube leads to a gold-plated silver canula. This canula is inserted through a phlebotomy opening into a vein just above the bend in the elbow. The vein is ligated distal to the insertion, and a ligature of umbilical tape is passed around the canula in the vein, held in situ by a shoulder on the canula. The wound is closed and a sterile dressing applied. The rubber tubing is fixed to the arm by strips of adhesive, permitting the patient to move the arm at will during the administration. The use of two thermos bottles permits the employment of one while the other is being filled. The thermos feature keeps the solution at a constant temperature.

It is noteworthy that in no case so treated has there been any reaction or other ill effects of the glucose administration, and further that the desired results have been obtained in every instance.

If the further results of this method prove as desirable as those already obtained, it will broaden the field of usefulness of glucose therapy, especially in the treatment of those severe toxemias of early pregnancy where other less intensive methods fail.

SUMMARY

From our observations enumerated herein, the following points might be summarized:

1. In the early toxemias of pregnancy, where the disturbance is of metabolic origin, the employment of glucose is probably our best single therapeutic agent.

2. In those early toxemias of renal origin, glucose is of value as a supportive and detoxicant, but does not relieve the hyperemesis as completely as in the toxemias of primary carbohydrate deficiency.

3. In the late toxemias, glucose is effective in lowering blood-pressure, neutralizing the toxin and lessening the number of eclamptic convulsions.

4. Recent experiments with the continuous drip method of the intravenous use of glucose tend to show that larger quantities of this agent can be used, and with more beneficial results, than was formerly supposed.

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INTRAPERITONEAL INFUSION & AUTOTRANSFUSION IN ABDOMINAL SURGERY*

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THE importance of an adequate supply of fluids after operation is generally recognized even by surgeons who still dehydrate their patients by preoperative catharsis and restriction of water intake. The use of the Murphy drip, hypodermoclysis and intravenous infusions is almost universal. In all, or nearly all clean abdominal operations these procedures are unnecessary annoyances to the patient, the surgeon and the nurses.

As shown by Wegner, absorption from the peritoneal cavity is so rapid that the injection of potassium cyanide causes death almost instantly, just as if it were injected into the blood stream. Normal salt solution is absorbed directly into the blood stream. Dandy and Rowntree[1] proved this by injecting a solution containing phenolsulphonephthalein as an indicator. The dye appeared in the urine in six minutes and in the lymph only after thirty-seven minutes. In an hour, 56.4 per cent of the dye was regained from the urine, while less than 0.1 per cent appeared in the thoracic duct. Clinical confirmation of this work is described below.

Why not utilize the peritoneum to furnish the patient with a continuous supply of water during the time when he needs it most? There is no limitation to the amount that can be absorbed except the capacity of the abdominal cavity. The technic is simple. After the last peritoneal suture has been inserted and before it has been tied, a catheter or small enterostomy rube is inserted and normal saline solution at 100° or 104°F. is poured by means of a funnel until the solution begins to run out around the tube. In the average case about 1 liter is used but after the removal of a large tumor, much larger amounts are taken.

The benefit of this procedure is twofold. It immediately overcomes the chilling of the peritoneum which is unavoidable in any extensive operation and it assures a constant intake of water for the six or eight hours during which it is needed most. Whether it prevents adhesions is debatable. The striking absence of postoperative thirst would justify the use of the method regardless of the general benefit to the patient. Since adopting this method, we have discontinued the use of the Murphy drip except where the intraperitoneal infusion is contraindicated by the presence of pus or by drainage of the wound.

The power of the peritoneum to absorb fluids as shown by animal experiment has already been mentioned. Clinically, we have checked it by the following experiment:

To 1 liter of normal saline solution, we have added 1 c.c. of standard solution of phenolsulphonephthalein solution. The urine has been collected by catheter at intervals of one, two and six hours and quantitatively tested. The average percentages of excretion are:

Hours	Per Cent
1	14
2	37
6	59

Since these figures are dependent not only on the rate of absorption but on the rate of renal excretion, it is obvious that a considerable degree of absorption begins immediately and the greater part of the solution has entered the circulation during the first six hours.

INTRAPERITONEAL AUTOTRANSFUSION

Many reports have appeared in recent years of the intravenous infusion of citrated blood taken from the abdomen,

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especially in cases of ectopic pregnancy. This is a logical procedure, especially when the abdomen is full of fresh unclotted blood from recent hemorrhage, whether from ectopic pregnancy or ruptured liver or spleen. It is much simpler and apparently as effective to citrate the blood and return it to the peritoneal cavity with the routine saline at the conclusion of the operation. We have done this particularly when hysterectomy was performed for bleeding fibroids. These patients are frequently anemic and it is desirable to save them from further loss of blood. Especially when dense adhesions must be separated, a good deal of bleeding is often unavoidable. Lesage[2] found in experimental animals that untreated blood is absorbed rapidly after three hours. Bizzozero and Golgi,[3] using defibrinated blood, found an increase in the number of red cells twenty minutes after injection. The maximum increase came after about two days. Hertzler[4] says, "The absorption of non-coagulated blood seems to be similar to the absorption of isotonic solutions in general."

By means of the ordinary Pool suction tube, free blood is aspirated into a sterile bottle containing citrate solution. A basin containing citrated saline solution is also placed conveniently so that an assistant can rinse in it each sponge as it becomes soaked with blood. At the conclusion of the operation, the citrated blood in the bottle and the saline-blood mixture in the basin are strained through gauze and warm saline is added to bring the volume up to 1000 c.c. and the temperature up to 100°F. The mixture is then introduced as in the routine saline infusion previously described. We have been doing this for nearly three years and have never seen the slightest ill effect. Many pediatrists have used the intraperitoneal route in administering citrated blood to infants, apparently with complete satisfaction. This seems to answer the theoretical objection that blood is a foreign body in the peritoneal cavity.

When intravenous autotransfusion is used every precaution is taken to prevent breaking up the red cells. This is emphasized by Farrar,[5] who says: "Care was taken not to stir or shake the blood to avoid injury to the red cells." Clots must be eliminated. An extra assistant is necessary.

In the intraperitoneal method, a few clots do no harm. Ruptured red cells are absorbed and the hemoglobin utilized. The blood-soaked sponges may be squeezed and even rinsed out in the citrate solution, so that the greatest possible salvage is effected. No extra assistant is required. If no suction is available, the greater part of the blood can still be saved by sponging or ladling it from the abdomen.

In the event of severe hemorrhage into the abdominal cavity, whether it occurs before or during operation, it is not good surgery to throw away the blood. This method of returning it to the patient is simple, safe and effective. It is a conservative measure which is worthy of more general use.

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THE PORGES-POLLATSCHEK SKIN TEST FOR PREGNANCY*

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A SIMPLE and yet reliable skin test would be highly desirable and of vast importance in the diagnosis of early pregnancy. The importance of such a test is particularly obvious in post partum cases where a slight increase in the size of the uterus occurring together with a lactation amenorrhea must be ruled out as signs of pregnancy. Its value is equally apparent in the diagnosis of a gestation at the premenopausal age. In cases of young girls of irregular menses and also of recognized social status, a skin test for pregnancy would be invaluable since it would eliminate a highly embarrassing and perhaps a presumptuous pelvic examination. Even in legal cases wherein the history must be disregarded and pregnancy diagnosed early, such a test would be of vast assistance and, last but not least, in instances where the physician is called in to differentiate between an ectopic and some non-surgical condition.

The Ascheim-Zondeck test for pregnancy might well be relied upon in all such conditions except in an ectopic gestation; it is very reliable since it gives 95 per cent to 98 per cent correct results in the hands of an experienced man. However, one must have facilities for keeping mice. This is a very difficult task for the following reasons: they must be virgins of a certain weight and age, and at that age, sex is hard to determine; occasionally they kill each other; frequently they die before injection; many die from meningitis or from toxic urine. Moreover the urine of a patient receiving antiluetic treatment kills the mice within forty-eight hours. And finally, it takes almost a week to make a diagnosis, thus rendering this task practically useless in ectopic gestation.

In the Vienna letter to the *Journal of the American Medical Association* on August 17, 1929, a simple and yet reliable skin test eliminating these objections was briefly mentioned. Dr. Otto Pollatschek and Dr. R. Porges of Vienna were injecting 0.2 c.c. of the hormone of the anterior lobe of the hypophysis intracutaneously. "If the woman is not pregnant, a distinct red circle, about an inch in diameter, is formed after a few hours at the sight of the injection, and remains visible for twenty-four to thirty-six hours. In pregnant women, there is no reaction." This was explained as a specific skin reaction which was an entirely new observation for hormones, and as analogous to the Schick, Dick and Pirquet test for infectious toxins.

Not convinced by the explanation of the mechanism involved, but interested in checking up the results of the European investigators, I obtained permission from the heads of the various departments of the Kings County Hospital to conduct this experiment. The only preparation of anterior pituitary hormone available in the United States was antuitrin. Later I was able to procure prolan, the preparation that the Viennese physicians used.

In order to avoid drawing unwarranted conclusions, I chose a series of over 500 consecutive cases. Of this original number, 50 were excluded because of failure to return for a reading. I discarded an additional 100 in order to thoroughly familiarize myself with the procedure and to eliminate trauma as a possible factor. Thirty more cases were thrown out due to the intense pigmentation of the skin. However, the greatest factor against this test was the indefiniteness of the results, which made us disregard 150 cases. The skin reactions were of such a nature, in this latter group, that 2 or 3 observers could not agree on the readings. Of the total number of 524 cases, only 184 remained, which are tabulated as follows:

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TABLE I

Condition	Total Number (Antuitrin)	Results
Males	6	5+, 1−
Menopause	1	1−
Postpartum	8	5+, 3+
Menstruating	1	1+
Pregnant	22	5+, 17−
Non-pregnant	28	18+, 10−
Inevitable abortion	1	1−
Ruptured ectopic	1	1−
Incomplete abortion	1	1±
	69	

TABLE II

Condition	Total Number (Prolan)	Results
Males	4	4+
Menopause	11	1+, 9−, 1±
Postpartum	13	3+, 7−, 3±
Menstruating	5	3+, 2−
Pregnant	36	12+, 24−
Non-pregnant	46	28+, 10−, 8±
	115	

The results obtained by the writer as just indicated lead to only one obvious conclusion: namely, that the so-called Porges-Pollatschek skin test for pregnancy is neither definite nor reliable. Subsequently, the writer's findings were confirmed by the very authors of this so-called test, for towards the latter part of the investigation, a communication was received from Dr. Porges himself, wherein he admitted that as his series of tests increased his percentage of error ran as high as 18 per cent. Obviously, this was quite a radical departure from the earlier assertions regarding the absolute reliability of this test, for originally it was claimed that the test had been proved correct in all but one instance, that of a woman who had a hypophyseal tumor.[1]

In conclusion, the writer would like to express his gratitude for the kind assistance and cooperation rendered him by Drs. Philip Smith and Raphael Kurzrock of Manhatten and Dr. M. Walzer of Brooklyn, but for whose helpful suggestions many of the obstacles which confronted him could hardly have been overcome.

[1] Further confirmation of the writer's findings has since appeared in the *Zentralbl. f. Gynak.*, 53 Jahrg. 16, Nov. 1929, No. 46, p. 1920, wherein Dr. Alfred Deutsch of Vienna, after a series of similar experiments, announced conclusions coinciding with those of the writer.

THE SURGICAL TREATMENT OF UTERINE FIBROMYOMA*

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UTERINE fibromyoma is the most common nonmalignant new growth occurring in the human body and relatively speaking, constitutes the most frequent pathological condition the gynecologist is called upon to treat. In a personal series of 416 pelvic operations taken chronologically, the writer found the presence of the ordinary lobular levicellular fibromyoma of the uterus in 68 cases or 16.3 per cent. All the patients were white women with the exception of one. These findings are considerably higher than those of Hofmeier and Essen Moler who estimate that they constitute from 4.3 to 4.7 per cent of all gynecological conditions. However, they are within fair keeping with those of Lynch's[1] and Frank's[2] most thorough accumulative data which shows, varying with different investigators, that 20 to 30 per cent white and 30 to 60 per cent colored women are prone to the development of uterine myomas, the fastigium being reached between thirty and fifty years of age.

There is no questioning the fact that thousands of symptomless uterine fibromata go unrecognized and that many are discovered accidentally. While a few unauthenticated cases have been reported as occurring before puberty, it must be recognized purely and simply as a disease of the child-bearing period of a woman's life, receiving its stimulus for development as a result of congestion incident to menstruation, confirmed in the fact that with the establishment of the menopause, the tumors tend to atrophy and degeneration.

ETIOLOGY

Many theories have been offered as to the probable origin of fibromyomas, the most feasible of which is that expounded by Roesger, Gottschalk and others that they develop from the musculature of small arteries.[3] However, not any border on a hypothesis. The most interesting singular fact is that histologically the tumors consist of the same connective and non-striated muscle tissues as constitute the normal uterine substance and differ only in their structural arrangement.

IRRADIATION

Soon following the discovery of radium by Madam Curie in 1898 and improved *x*-ray technic, there was a tremendous change in the treatment of uterine fibroids and it seemed for a time the surgical treatment would become extinct. However, today, in the vast majority of cases it must be acknowledged as the most important and trustworthy of all known methods. It is not the intention of the writer to belittle the value of radium and *x*-ray in the treatment of uterine myomas, although he is of the opinion they are overestimated by the enthusiasm of many roentgenologists and radium therapeutists. Obviously, W. J. Mayo clearly stated the sentiment of the surgical profession when he said "Radium must justly be considered in selected cases as a competitor of hysterectomy but it has no comparative standing in cases suited for myomectomy."

CLASSIFICATION OF INDICATIONS FOR OPERATION

In the presence of uterine myoma there are certain definite indications under which surgical intervention should be instituted unless precluded by constitutional diseases or obesity, the presence of which would necessitate a hazardous risk. For the purpose of description they have been

* Read before the Academy of Medicine, Columbus, Ohio, October 14, 1929.

classified with relation to their importance as follows:

1. Tumors with benign or malignant degenerative changes which Noble has classified as follows:[4]

CLASSIFICATION OF DEGENERATIVE CHANGES

Form of Change	No. Cases	Per Cent
Hyaline	72	3.1
Hyaline with calcareous infiltration	8	0.3
Calcareous	39	1.7
Myxomatous	89	3.4
Cystic	58	2.5
Hemorrhagic	13	0.57
Necrosis	119	4.7
Fatty degeneration	7	0.25
Edema	17	0.74
Sarcoma	34	1.4
Carcinoma corpores	42	1.8
Carcinoma cervix	16	0.7

He finally concluded that these degenerative changes occurred in 62 per cent of fibroma of the uterus. It is unnecessary to mention the importance of early recognition and radical operation in the presence of the last two mentioned conditions, namely sarcoma and carcinoma. Even as early as 1921, Essen Moeller[5] reported 22 malignancies in 700 fibroid cases, 6 of which were cancer and 16 sarcoma. That sarcomatous degeneration occurs relatively more frequently than is commonly believed by the general medical profession has been proved by Winters'[6] most thorough and definite microscopical findings of 4 per cent. The frequency of carcinoma, which in itself is not a definite degenerative process as it must originate from epithelium, has been proved by Weibel[7] who reported a series of 1000 uterine myomas in which carcinoma of the fundus was found in 20 cases and Noble[8] who states that cancer was present in 2.8 per cent of a series of 4880 cases of fibroids collected by him. The cervix was involved in 1.29 per cent and the fundus in 1.05 per cent. Note the reversal of the location of the tumors to that found under ordinary conditions. In the writer's series of 68 uterine fibroids, 3 cases were complicated by carcinoma of the fundus and 1 by carcinoma of the cervix.

2. Tumors accompanied by inflammation frequently subsequent to degenerative changes involving the tumor itself or infection or disease of the appendages. Cullens, Popow, Virchow, Tait, McDonald and Webster are of the opinion that 50 per cent of all uterine fibroids are accompanied by inflammation of the adnexa. However, as in the case of the hen and the egg, no one seems prone to state definitely whether the tumor resulted from the appendageal inflammation or the appendageal inflammation resulted from the presence and growth of the fibroid. Nevertheless, the presence of the inflammation in association with fibroids is a definite indication for surgical intervention and surgical intervention only, not radium or *x*-ray.

3. Tumors accompanied by pressure symptoms resulting from impingment on the bladder, ureters, and rectum characterized by irritability of the bladder with frequent, urgent, and painful urination and not infrequently urinary retention, which unrelieved, ultimately terminate in cystitis with bladder wall distention and thickening, hydroureter and hydronephrosis with final permanent destruction of the normal kidney substance. Pressure upon the rectum frequently results in constipation, autointoxication and anemia. The tumor or tumors are usually wedged and firmly fixed in the pelvis; however, small intraligamentous tumors are frequently of no little source of disturbance.

4. Tumors 15 cm. in diameter equal to four months' pregnancy or larger should be removed surgically especially in women under forty years of age as irradiation in sufficient amount to cause an appreciable shrinkage in the size of the tumor would result in irreparable damage to the ovaries. In fact, opinion is rapidly becoming established that the effect of irradiation on uterine fibroids is secondary to its effect on the ovaries. Masson states: "Apparently there is little or no decrease in the

size of a tumor after treatment by roentgen rays or radium unless the doses are powerful enough to precipitate the menopause."[8]

5. Tumors present in sterile women of suitable age with a desire to bear children when other conditions productive of sterility have been eliminated should be removed surgically. Mayo reports 23 pregnancies following myomectomy in 151 married women and Stacy, 28 pregnancies in 203 myomectomized women.

6. Tumors with relation to their position which may be classed as pedunculated and submucous in type, all doubtful cases and rapidly growing tumors, especially those accompanied by hemorrhage unrelieved by other known means.

TYPES OF OPERATIVE PROCEDURE

The surgical procedure necessarily resolves itself into two definite plans, myomectomy, conservative in type, and hysterectomy, radical in type, each of which has proved of equal safety relative to mortality. At the Mayo Clinic extending over a period of five years, 259 myomectomies had been performed with 2 deaths (0.77 per cent) and 1643 hysterectomies with 31 deaths (1.88 per cent).[9] In 1926 under the caption of "The Safety of Hysterectomy" the writer reported a personal series of 182 hysterectomies with a mortality of 1.07 per cent. Since that report, 74 additional hysterectomies have been performed, increasing the total to 256 with 9 deaths or a mortality rate of 3.51 per cent. An increase in mortality has been noted which is accounted for in the fact that with increasing experience, a feeling for more conservatism in pelvic surgery has developed which naturally relegates the less critical cases to myomectomy, leaving the more critical cases for hysterectomy. Statistics show death following either operation to have resulted from accidental factors in the majority of cases, pulmonary embolism contributing to over 50 per cent of the cases.

Myomectomy like hysterectomy has certain definite indications for its adoption. However, each case predisposes certain personal status in regard to age, occupation, social and financial position and the desire to bear children. When these deductions have been made, myomectomy may be said to be the operation of choice in the majority of patients under thirty-eight years of age with single or several isolated tumors located in such a position in the uterus that they can be readily shelled out without unreasonable damage to the uterine substance. Especially does this apply to isolated sessile and pedunculated tumors as well as many of the interstitial varieties. The hope of pregnancy and desire to conserve the menstrual function call for conservatism. Bonney successfully removed from the uterus 30 fibroids in one patient by myomectomy. Kelly successfully removed 9 tumors through separate incisions on one patient and 12 tumors in another, and twice removed tumors as large as a man's head by myomectomy.

METHOD OF HYSTERECTOMY

Hysterectomy solicits three methods of procedure, namely, subtotal, total and vaginal, each of which should be determined by definite indications. The uterus should be removed in part in the presence of all fibroid tumors accompanied by hemorrhage or profuse discharge uncontrollable by other means. Total hysterectomy should be performed when definite evidence signifies involvement of the cervix. That total hysterectomy can be performed by competent surgeons as safely as subtotal has been proved by the data of many operators. In a personal series of 256 hysterectomies there were 159 total hysterectomies with 6 deaths (3.64 per cent), 96 subtotal hysterectomies with 3 deaths (3.12 per cent), and one vaginal without mortality.

Upon sufficient evidence of malignant degeneration but one procedure is left to choose from and that is panhysterectomy. At the Mayo Clinic over a period of five years 16 cases of carcinoma of the cervix were accumulated from three to

fifteen years following subtotal hysterectomy for non-malignant growths and 13 cases in which it was impossible to determine the existence of malignancy at the time supravaginal hysterectomy had been performed. Subtotal hysterectomy should be the choice, once the involvement of the cervix has been ruled out, in all cases of uterine myoma complicated by inflammation of the appendages, especially when as a result of severing adhesions the organ would be denuded to such an extent that further adhesions in the pelvis would be inevitable, and in all cases in which removal of the fibroid by myomectomy would necessitate an appreciable sacrifice of uterine substance.

SUMMARY

It is the writer's conclusions that the surgical treatment of fibroids has been too radical; that sound surgical judgment must be exercised in determining the choice of operation lest one become ultra-conservative which leads to unsatisfactory results; that the value of irradiation has been overestimated and finally, it must be remembered that a patient with fibroids is being operated on and not alone a uterus with fibroids.

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AN IMPROVED AIR INJECTION APPARATUS FOR THE INFLATION OF JOINTS*

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RECENTLY there has been a great deal of work done on the inflation of joints, either of the chronic arthritic or acute purulent types. In the main, this has been carried out by means of the syringe method or by means of more or less cumbersome implements; therefore it was with this idea in mind that an improved method was sought. The first personal attempt was carried out on the Bone and Joint Service of the Boston City Hospital in 1928, and there the cases chosen for the inflation were mainly of the acute gonorrheal type. The results in these were quite encouraging, but it is not the author's idea in writing this paper to bring out the clinical data established in those cases. The purpose of this paper is to present to the profession a very compact and efficient apparatus for the inflation of joints by air.

The advantage of the improved apparatus is, of course, to allow for complete aseptic aspiration of the diseased joint, and secondly to have an absolutely aseptic method of inflating the joint with sterile air, at the desired pressure.

The complete set as shown in Figure 1 consists of a mercury manometer, a sterilizing chamber, a detachable rubber connection, one end of which attaches to the exhaust of the sterilizing chamber, the other end fitted with a two-way adaptor. The adaptor will accomodate the syringe and aspirating needle. Figure 2 shows the detachable portion of the set and is seen to consist of the rubber connection, adaptor, syringe, needle and release valve. This portion is boiled as often as it is used, and if necessary several of these parts can be kept on hand in sterile packets all ready for use.

The manometer, sterilizing chamber and pressure bulb are mounted on a small detachable metal stand. All parts can easily be taken down and will fit into a very compact case.

Principle of Operation. The principle consists in delivering air to the sterilizing chamber which is constantly governed by the manometric pressure as indicated on the mercury column. The air is bubbled through the sterilizing solution which completely precludes the danger of carrying any particles of dust or contaminated material over into the joint. The exhaust tube of the sterilizing chamber therefore supplies the air in pure form to the sterile rubber tubing and attachment. The pressure is constantly governed and easily controlled by the bulb attached to the manometer. The sterilizing solution used is 70 per cent alcohol.

Technic. The joint to be inflated is scrubbed, shaved and painted with iodine or other suitable skin antiseptic. Sterile sheets are draped around the area to be used and the sterilized unit is then attached to the exhaust of the sterilizing chamber. The syringe and aspirating needle are attached to the threeway adaptor and with the skin previously anesthetized with the aid of novocaine or ethyl chloride, the needle is inserted into the joint cavity. The fluid, if there be any, is then aspirated by means of a 20 c.c. syringe and the specimen kept in a sterile tube for bacteriological examination. When all the fluid has been removed, the two-way cock is turned, thereby opening the channel between the sterilizing air chamber and the aspirating needle, and cutting off the path to the syringe. The air is then slowly forced into the joint by pressure on the bulb in much the same manner as in taking a blood pressure. The joint space is then seen

* Submitted for publication February 17, 1930.

gradually expanding and depending upon the amount of pressure used, will be either soft or quite firm to palpation. Generally

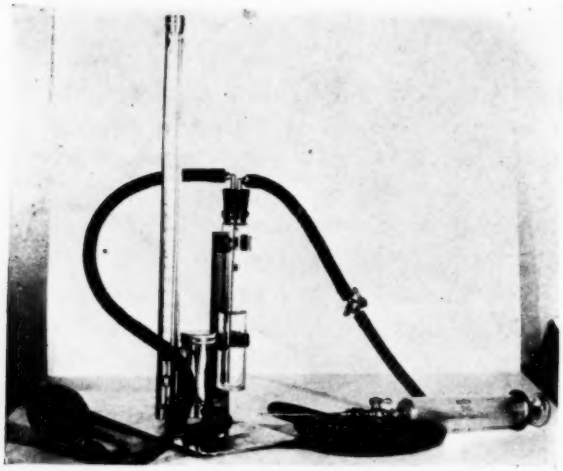

Fig. 1. Complete set assembled and ready for use. Sterilizing chamber and manometer attached to a demountable metal stand. All parts fit into a small compact case.

speaking, 40 to 50 mm. of mercury pressure will be easily tolerated by the average

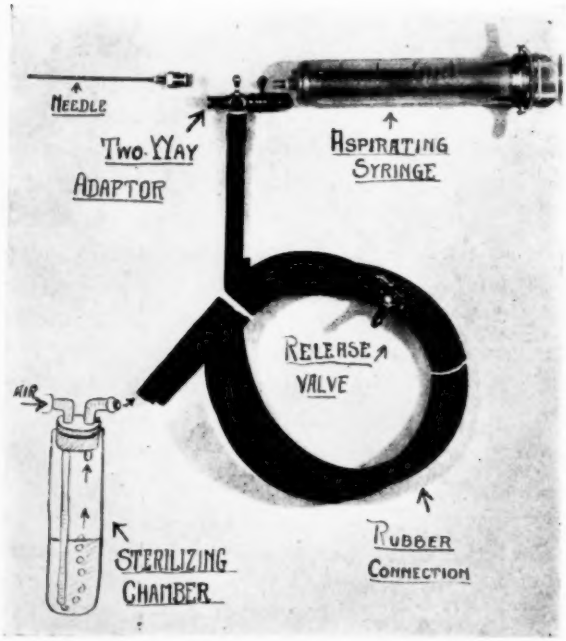

Fig. 2. Boilable parts of set. Release valve allows immediate release of injected air, if so desired.

patient without discomfort. It does not seem that greater pressures are necessary, as under these conditions it will be seen that only the resistant, thickened or adherent synovial membrane will remain in contact with the adjacent tissues. When

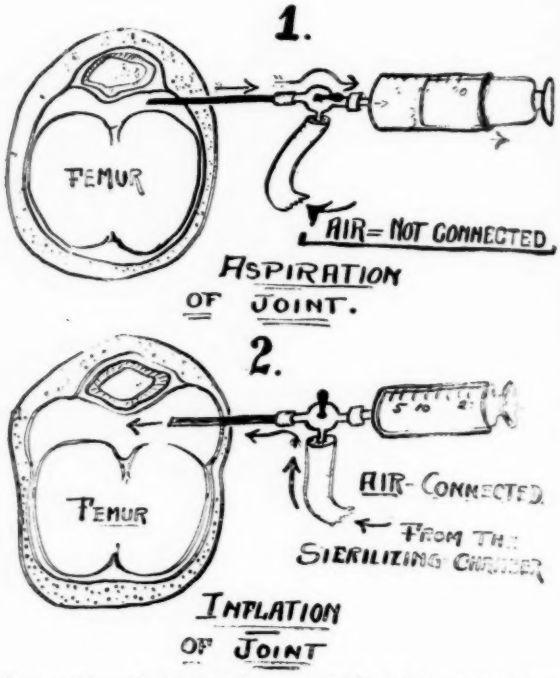

Fig. 3. Drawings showing needle in joint space. No. 1 shows aspiration to remove fluid, and ascertain that needle is not in blood sinus.
No. 2 shows injection of sterile air, with synovial membrane distending.

the desired pressure has been attained, the needle is withdrawn and the punctured wound painted with collodion. An injection usually suffices to keep the surfaces apart for a period of from three to seven days after which time it can be repeated. If another joint is to be injected at the same time, the syringe and aspirating needle together with the rubber connection may be reboiled or another set kept on hand for immediate use. *It is very essential before injecting air that the needle is not in a blood vessel or sinus, as serious results may ensue if air under pressure is injected into open veins.*

Summary. The author has personally used this method on the following types of joint condition:

1. Chronic arthritis with effusion.
2. Acute gonorrheal arthritis.
3. Acute infectious arthritis.
4. Synovial tuberculosis.

5. Hemophiliac joint (1 case only).

6. Postoperative tuberculous synovitis with synovectomy (1 case only).

7. Suggested use on joints immobilized in plaster casts over long periods of time to try to prevent some of the troublesome joint stiffness seen in these cases, reference being made to cases with fractured femurs, etc., in plaster spicas.

The results of these cases seem very promising and interesting, and the data will form the basis of a later paper. It is felt that in presenting this simplified set, that a great many more of the profession will realize the adaptability of an accurate and simple apparatus in the growing practice of producing pneumoarthrosis in certain pathological joint conditions. The author sincerely hopes that some of the men who are particularly interested in this line of work will offer any suggestion or criticisms they may deem advisable.

The author wishes to express his gratitude to Dr. E. A. Sommer of Portland, Oregon, and to Drs. W. B. Coley, Arthur Krida, Bradley Coley and J. McWhorter, for their kind suggestions and help in this work.

CASE REPORTS

CHRONIC PANOSTITIS*

INVOLVING ATLAS AXIS AND THIRD CERVICAL VERTEBRA

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INASMUCH as panostitis of the upper cervical vertebrae is uncommon this report of a fatal case should prove of interest.

A thirty-seven-year-old female, married, had been admitted to the hospital on four occasions within a period of about twenty months.

The only previous noteworthy illnesses had been chicken-pox in childhood and influenza in adult life.

March 26, 1928, first admission to hospital, diagnosis: Neurotic with mitral disease. Throat: "pillars injected, red, tonsils are red, congested, appear infected." Basal metabolism rate, +13. Urine, negative. Wassermann, reaction negative. White blood corpuscles 13, 150; polymorphonuclear 76 per cent, small mononuclears 13 per cent, large mononuclears 8 per cent. Sugar tolerance fasting 102; first hour 308, leucocytes second hour 250, third hour 220, suggesting hyperthyroidism. Gastrointestinal series, negative. Gall bladder normal.

August 14, 1928, second admission, diagnosis: Thyrotoxicosis (infective in origin). Pharynx appeared injected, also left anterior pillar. Throat shows small atrophic tonsils from which cheesy exudate is easily expressed. "If thyroid disturbance exists it is probably on an infective basis from tonsils and gums." B.M.R. +8.

September 8, 1928, consultation and examination by Ear Nose and Throat Department revealed left tonsil submerged, anterior pillars inflamed, considerable caseous material expressed from left tonsil. B.M.R. −6. Impression: "chronic infected tonsils."

October 4, 1928, Tonsillectomy under local anesthesia with much improvement in patient's condition. Weight on admission 91½ lb., on discharge 94 lb. Hemoglobin 75 per cent, R.B.C. 3,890,000. W.B.C. 7200, P.M.N.L. 68 per cent, S.M. 30 per cent, L.M. 2 per cent. Throat culture, staph. and strep. Sugar tolerance fasting 120: I, 200, II, 205, III, 190.

May 13, 1929, seen in Out-Patient Department, Ear Nose and Throat Clinic, with an acute coryza; complaining of a burning sensation in posterior pharynx. There was some injection, with irritation of the mucous membrane. Lymphoid tissue in upper pole, left tonsillar fossa. Aseptic treatment directed to nose, none to throat.

July 18, 1929, throat painted with silver nitrate 2 per cent. Complained of being afraid to eat because of a choking sensation. Impression appears to be hyperthyroidism, though B.M.R. has always been within normal limits.

July 25, 1929, seen at home suffering with acute lateral pharyngitis which gradually localized with abscess formation over right posterior pillar. Incision with release of pus. Remained in bed at home for one week. Neck began to feel stiff, and throat not so sore.

August 8, 1929, seen in Out-Patient Department, Ear Nose and Throat Clinic, complaining of ears aching, difficulty in swallowing at times, neck feeling rigid. Appears to have lost great deal of weight. There is a discharging opening in right posterior pillar which is much swollen. Neck is held rigidly, some tenderness over muscular insertions. Temperature, 98F°. Pulse, 104. Throat culture: Strep. predominating; also Putrifecans. *x*-ray of cervical vertebrae with report of "No radiographic evidences of pathology."

August 11, 1929, third admission, diagnosis: Retropharyngeal focal infection giving rise to cervical myositis and fibrositis. Urine, negative. R.B.C. 3,710,000. W.B.C. 10,000; P.M.N.L. 75 per cent, S.M. 14 per cent, L.M. 9 per cent, T.R. 1 per cent, E. 1 per cent. Throat culture, Strep.

August 13, 1929, examination revealed an

* Submitted for publication, February 26, 1930

opening surrounded by pale granulations on the right posterior pharyngeal wall in region of the posterior pillar, discharging thick greenish-yellow pus. The area was swollen, with some stiffness of the neck especially on backward movement; tenderness over back of neck, more marked on left side. Cervical glands not palpable. No tenderness over spine. Impression: "Post-pharyngeal space abscess; vertebral caries?"

August 15, 1929, under local anesthesia, the pharyngeal opening was widened and granulations curretted. Probe went back to spine, but no bare bone was felt.

August 22, 1929, injection around opening a little smoother, with paler cheesy pus still discharging from the sinus. Neck rigidity is perhaps a little less.

August 24, 1929, Esophagoscopy: nothing below cricoid; edema above cricoid with narrowing of esophagus.

September 7, 1929, discharged from hospital to return to Out-Patient Department for diathermy and observation.

September 14, 1929, seen in Out-Patient Department, improving, being treated by physiotherapy.

September 19, 1929, an *x*-ray with probe inserted in fistulous tract revealed "Rather marked destruction of the upper anterior portion of the body of the third cervical vertebra which was not present on previous examination, (August 8, 1929). The probe inserted through the fistulous tract seen to extend down to this necrotic portion."

September 21, 1929, transferred to Orthopedic Clinic. On examination patient appeared nervous, complained of having a choking sensation for past two years. Presence of a chronic discharging abscess and sinus in right retropharyngeal space would indicate that a long standing pathology exists in that region. The probability of bony involvement was immediately suspected. All efforts at movement of head, painful and very limited. Some muscular rigidity of the anterior muscle groups of the neck. Head held very guardedly. Loss of weight apparent for past two years. Immediate hospitalization recommended.

September 24, 1929, *x*-ray of cervical vertebrae revealed necrosis previously noted in the third cervical vertebra further advanced with some beginning necrosis in the lower portion of the second cervical vertebra.

When she was referred to the Orthopedic Department the diagnosis of cervical tuberculosis was made clinically. It was felt that regardless of the type of bone disease the

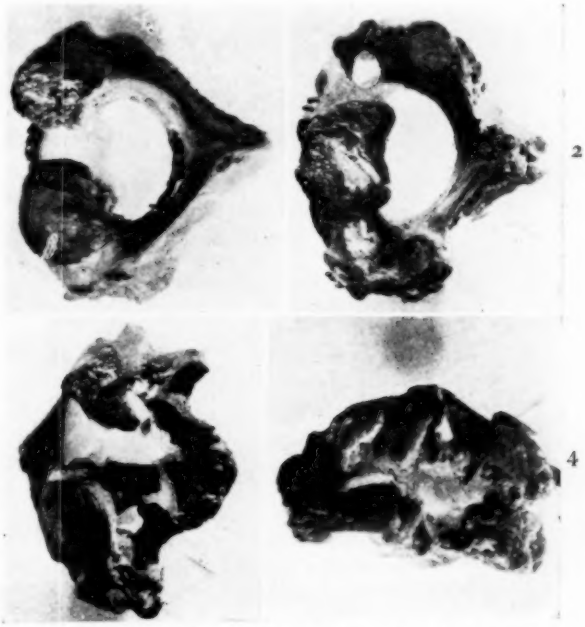

Fig. 1. Looking down upon atlas, showing marked destruction of anterior arch and extensive erosion of right superior articular surface.
Fig. 2. Looking up upon base of axis, showing erosions.
Fig. 3. Anterior view, showing destruction of odontoid process and extensive destruction of atlas and axis.
Fig. 4. Posterior view, showing extensive erosion and ossification between laminae of atlas and axis.

treatment of primary importance would be recumbency with fixation and temporary moderate traction. Because of the many neurological symptoms manifested, e.g.: exophthalmus, nystagmus, light flashes, spots before the eyes, tremor, as well as choking sensation following swallowing of food, enlargement over thyroid and the general imbalance of the sensory nervous system, a consultation was requested with the Neurological Department. The diagnosis, with remarks follow: "T.B. of atlas (Runge? syndrome), T.B. of thyroid (?) or hyperthyroidism. These conditions are usually fatal. Outlook very grave. Only treatment is complete fixation of the head. Morphine sulphate to be used freely." Hg. 75 per cent, R.B.C. 3,950,000. W.B.C. 11,300: P.M.N.L. 78 per cent, S.M. 21 per cent, L.M. 1 per cent. Sputum and throat culture, negative for tubercle bacilli.

During the patient's recumbency the head fixation with traction was carried to the point of tolerance. There were times when it became

necessary to relieve weight suspension because of aggravation of pain which this treatment occasioned. However, after recumbency with a varied fixed policy of head traction for about six weeks, during which time a normal temperature was established and the general condition of the patient improved, a semi-soft collar was decided upon. To apply this apparatus was a delicate procedure in this instance, because of the danger of disturbance to the suppurating process and a flare up of the bony pathology.

Fig. 5. Film taken August 8, 1929, showing no evidence of pathology.

Fig. 6. Film taken September 19, 1929. Probe inserted through fistulous tract is seen to extend down to necrotic third cervical vertebra.

October 17, 1929 a snug and highly fitted collar of felt with plaster-of-Paris reinforcement was placed about the neck. Close upon the application of this collar, although the patient complained about its being tight and limiting the motions of the chin, she admitted she was more comfortable. The general symptoms complained of, pains about the head, vertigo, flashes, spots and blurring of vision, difficulty in swallowing, swelling and pain about the ears, etc., gradually diminished. The patient became more cheerful and began taking on weight. Two days after application of collar, she was able to sit up with support of a back-rest although vertigo, tinnitus and impaired vision were still fleeting disturbing factors.

October 25, 1929, began getting out of bed on wheelchair.

October 29, 1929 she was able to walk about with assistance and after a few days was walking unaided. Patient presented all evidences of feeling much more comfortable than on admission to the Service and plans for her going home soon were uppermost in her mind. During the course of this treatment patient was constantly swallowing the discharge from the fistulous retropharyngeal opening. She was instructed to expectorate this material whenever possible, and it was no difficulty at any time to obtain pus which was thick and very tenacious in character, sometimes yellowish green in color and often blood-streaked.

November 4, 1929, or thereabout, it was found that the collar support was becoming very loose, that the neck was much smaller, and that even the height of the collar was insufficient to hold the head in the original fixed position. Symptoms such as those complained about on admission began to reappear fleetingly and for increasingly longer periods. It could be seen that the collar was not holding the head sufficiently firm. Because of the increased mobility of the head the collar was elevated with a ½ in. thickness of piano felt which was placed between the lower portion of the collar and the base of the neck as a tem-

porary expedient, until a new collar could be applied. A gradual improvement in her general condition with temperature decreasing and pain diminishing was apparent.

November 12, 1929, or thereabout, a setback from her previously encouraging convalescence set in most rapidly. There was a sudden marked rise in temperature with correspondingly increased pulse and respiration rates. The face became flushed, patient complained of severe headache, pains in ears, dizziness and marked disturbances of vision.

November 14, 1929 in the evening patient developed an alarming condition. It was reported she had raised herself in bed and that the head fell back, as though snapped, when she was in the erect position. In all likelihood the odontoid process of the axis broke away at this time. (At post mortem the axis was found with this process unattached.) When seen by me on this evening the patient was in a comatose state, head firmly fixed, face flushed, pupils dilated and eyes staring vacantly. Her speech was of a mumbling muttering type except when aroused. She was able to give yes or no answers to questions and her sight was not entirely gone. The head was drawn backward, the knees and hips flexed. She gave the appearance of having some central nervous disturbance of serious character.

November 15, 1929 when seen in the morning her condition was grave. She appeared moribund. She had a vacant stare, eyes widely open, pupils dilated with no reflex response to light or accommodation. There was no response to questioning, she mumbled incoherently, upper and lower extremities were flexed, face flushed and expressionless. It was clear there was a marked central disturbance with involvement of the meninges.

November 16, 1929, patient expired.

An autopsy was performed by Dr. K. Yardumian, pathologist of Montefiore Hospital, and his report follows:

The body is that of a white female, about the age of forty, fairly well nourished and well developed. Rigor mortis well set; moderate lividity present at the dependent parts of the body. On general inspection, the neck was found to be surrounded by plaster-of-Paris cast. The head was covered by dark brown hair with a few gray ones in between. There was a decubitus at the posterior part of the occipital region, the size of a silver quarter. The pupils were evenly dilated. The cornea was slightly opaque. The ears and nose were grossly negative. The muscles of the neck were atrophic.

Fig. 7. Film taken September 25, 1929. Necrosis previously noted in third cervical vertebra is still present and beginning necrosis is now seen in lower portion of body of second cervical, and some rarefaction of body of first cervical vertebra.

Permission for autopsy was authorized to the head and limited portion of the neck.

The usual incision, from ear to ear, was made on the scalp. The cranium was sawed and the brain exposed. The meningeal vessels were dilated. There was a moderate increase of cerebrospinal fluid in the subarachnoidal space. The fluid looked rather clear and transparent. On further sectioning, at the posterior fossa of the base of the cranium and below the falx cerebellum, there was a great amount of turbid fluid. The inferior surface of the cerebellum and the pons at the medulla were covered by thick yellowish fibrinous exudate. Smears and cultures were taken from these respective places. The brain was removed as a whole and amputated below the medulla. The anterior margin of the foramen magnum looked eroded and rough and was covered with similar exudate. Posterior incision was made at the angles of the occiput and the neck, and the

first two vertebrae were removed (that is atlas and axis). The two bones were firmly united to each other. No motion could be elicited. The body of the atlas looked rough and rarefied. One condyle looked fairly normal; the other one was slightly eroded. The odontoid process of the axis was completely necrosed. The articular surface was destroyed and was very rough; portions were easily pulled away and ragged. It was rarefied and disintegrated. No evidence of thickening or healing process found. No caseation present. The body of the atlas showed similar destructive changes. The periosteum was denuded. The third cervical was palpated at its position, but not removed. The body of it was rough, necrotic and rarefied. Numerous smears and cultures were taken from those necrotic areas.

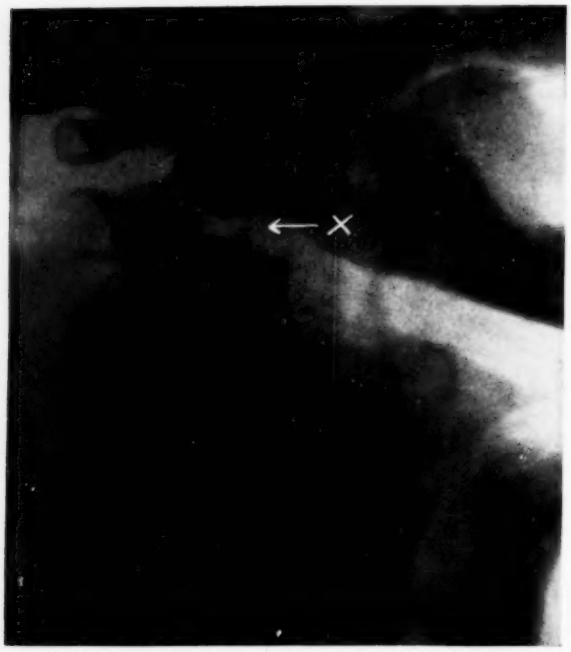

FIG. 8. Post-mortem *x*-ray. Further advance of necrosis of third and second cervical vertebrae, and also necrosis of first cervical.

Through the same incision, an attempt was made to remove pieces of thyroid gland. Extreme care was taken not to disfigure the body; therefore the incision was closed without further exploration.

Finger introduced through the mouth to nasopharynx at the superior posterior angle near to the center showed a sinus leading to the vertebrae of the neck. A large amount of purulent exudate ran out freely.

Microscopical Studies: Numerous smears were stained for acid-fast organisms and proved negative. Cultures from meningeal exudates from various necrosed bones proved a growth or organisms of which staphylococcus pyogenous aureus predominated. Other organisms were common flora of nasopharynx, such as diphtheroidal bacilli, few streptococci, pneumococci, etc.

Pieces of bones were removed from the odontoid process of the axis and the body of the atlas was decalcified in 10 per cent nitric acid solution, sectioned and stained.

The microscopical picture of the bone is as follows:

Marked rarefication and decalcification of the body; intense leucotic infiltration both polomononuclear and mononuclear type.

No attempt of regeneration and new bone formation is present.

No evidence of caseation of tubercular reaction seen. Also acid-fast stains disproved presence of any such organisms.

Diagnosis: Chronic panostitis involving bodies of the first, second, and third cervical vertebrae.

Terminal purulent meningitis involving the base of the cerebellum pons and medulla (staphylococcus pyogenes aureus predominating and few of the designated flora of the nasopharynx).

X-ray examinations and reports by Dr. Maurice F. Goldsmith, roentgenologist, Montefiore Hospital.

It is interesting to see how really far necrosis of the cervical vertebrae advanced in this case. An heroic effort had been made by Nature to overcome this condition and the inability to fully fix this effort at ankylosis resulted in a meningeal involvement. The first, second and third vertebrae were all involved in this extensive process of bony necrosis and the brain involvement certainly exceeded expectations.

An ostitis of any of the cervical vertebrae, more especially the upper cervicals, presents commonly a discouraging picture. This is further manifested after it has progressed into the chronic stage. An acute involvement of this type frequently responds favorably to treatment by free opening for drainage of the abscess, permitting same to repair from within outward. Fixation in such cases is mandatory, and is best obtained by head traction and weights at the outset to overcome spasm of the cervical musculature. These

muscles are in rigidity and spasm because of the effort of Nature to immobilize the vertebral bones involved. So long as drainage takes place from a sinus, whether retropharyngeal or postcervical, there remains nothing more to do beyond immobilization of bones of the diseased area. Further surgical interference is ill-advised. When the muscle spasm of the groups holding the head in rigidity has been overcome a proper fixation collar which rests directly upon the midclavicular region and base of neck is adequate support for the head. With such a collar the patient may be considered an ambulatory case. However, it should be realized that the swelling occasioned from the infiltration of such an inflammatory process and the infective materials in the soft structures about the involved vertebrae may cause considerable thickening of the neck. In such circumstances, what may appear as a tight-fitting collar at the outset will become very loose in the course of drainage of pus through an established sinus. The inflamed and infiltrated soft structures endeavor to assume their normal state. Therefore, the collar must again be adjusted for closer fitting from time to time to the neck of lessened thickness. Thus, there is further endeavor to prevent undue mobilization, with its consequent destruction from irritation, which would occasion a flare-up of the whole process with dire consequences.

In conclusion it is interesting to note that in cases of extensive necrotic cervical panostitis little remains to be done beyond fixation of the head and neck and expectancy from drainage obtained through any location whether retropharyngeal or postcervical. Ideally surface drainage is preferred to that of the oral route. Nothing can be expected from drainage alone. From fixation much may be hoped for even though drainage is not yet established. Then, too, there is always the question of doubt as to type of infection in the cervical region. It may be tubercular and operation for evacuation of pus would be contraindicated. Whereas, when abscess formation has established itself, it can be readily determined whether same is non-tubercular. Opening for drainage when imperative might take place in the retropharyngeal region, but most certainly should not be the point of election for same if the postcervical region is available.

The diagnosis of tuberculosis of the cervical vertebrae is not amiss in a case of this sort. One would almost invariably consider this form of infection first. However, in any type of panostitis, the treatment would always be the same, namely: fixation and drainage. We were not convinced up to the last point of demonstration at autopsy as to the type of organism underlying the initial phase of the case. When finally the Bacillus tuberculosis was eliminated and it was established that the infection was from without (oral cavity), in all probability of recent date, the case was considered of sufficient interest for presentation. The predominating organism in the lesion was, as noted in the pathological report, Staphylococcus pyogenes aureus.

An expression of gratitude is extended Drs. M. F. Goldsmith and K. Yardumian for their helpful cooperation in preparing this presentation.

ACUTE PANCREATITIS

WITH A REPORT OF TWELVE CASES*

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NEW YORK

UNDER this heading are included (1) acute hemorrhagic, (2) acute gangrenous and (3) acute suppurative pancreatitis. It is generally considered that these are merely various phases of the same disease and may follow one another or may occur concurrently in different parts of the pancreas in the same individual.

Acute pancreatitis is one of the tragic diseases of surgery. With sudden onset and rapid development it progresses all too frequently to a fatal outcome. Its etiology is obscure, its diagnosis difficult and its treatment unsatisfactory.

There has been much work done and many articles written on the various theories as to how infection reaches the pancreas. Deaver, Sweet, Judd and Mann have been the principal proponents of the lymphatic theory of infection, while Archibald and Kaufmann have carried out many experiments which would seem to show that infection takes place through the ducts. Superficially it would appear as if these two theories were diametrically opposed to each other and in fact their advocates seem, to some extent, to take such a view. Careful study of their various reports, however, does not necessarily lead to that conclusion. The adherents of the lymphatic theory are talking in the main about chronic pancreatitis which seems to be an entirely different condition and much more common than acute pancreatitis. That they are entirely different is pointed out by Sweet[1] who says "Chronic Pancreatitis equals infection, Acute Pancreatitis equals liberation of trypsin, by infection or trauma." Deaver[2] says "Extension of inflammation of the gall bladder mainly by way of the lymphatics gives rise to lymphadenitis along the course of the common duct and its lymphatics in the right free border of the gastrohepatic omentum, and to a peripancreatic lymphagitis and lymph adenitis with enlargement and hardening of the adjacent portion of the pancreas itself." He cites several cases where this occurred not only from an inflamed gall bladder but also following an acute inflammation of the appendix. But he makes no claim to having seen anything more acute than a lymphangitis of the pancreas following such conditions. In the same article Sweet,[2] after discussing from an embryological standpoint the various relations possible between the emptying of the common and pancreatic ducts says:

> Infection of regional lymph nodes might well cause a simple damming back of lymph with aseptic swelling of the interstitial tissue, later organization and increased interstitial tissue, which is precisely the picture of *chronic* pancreatitis. And Cases of chronic pancreatitis do not show clinical symptoms which might be ascribed to transitory attacks of limited acute pancreatitis with later transformation of the degenerated acini into sclerosed areas.

Mann's experiments as reported by Judd[3] consisted of injecting bile into the pancreas with a syringe at a pressure of 500 mm. or less, he having previously ascertained that the normal pressure of the liver and the contraction of the gall bladder combined did not exceed 350 mm.; and was not able to produce an acute pancreatitis. Archibald[4] doing the same experiment but using infected bile produced a typical necrosis of the pancreas. In summing up Judd[3] says:

> If Mann's conclusions regarding the bile factor in the etiology of pancreatitis are cor-

* Submitted for publication January 9, 1930.

rect, it would seem that, in most cases at least, we must search elsewhere for the cause. It will most likely be found, as Deaver says, in the extension of infection to the pancreas through the lymph channels. It is possible that infection in cases of cholecystitis changes the bile so that it readily causes trouble in the pancreas.

Nordmann[5] found that normal bile entered the pancreatic duct without doing any particular harm while infected bile caused acute pancreatitis. Mann and Giordano[6] and Judd[3] have shown that in only 3.5 to 4.5 per cent of bodies examined, obstruction at the ampulla of Vater either by a stone obstructing the ampulla or by spasm of the muscle of Oddi, would convert the common bile duct and the duct of Wirsung into one continuous passage and so permit passage of bile into the pancreas. If one accepts the theory of infection through the ducts this fact which has been used as an argument against the theory might tend to explain the relative rarity of acute pancreatitis. Kaufmann's[7] experiments consisted of infecting the lymphatics around the gall bladder, around the ducts and around the pancreas itself. He succeeded in causing a lymphangitis and a lymphadenitis and by contiguity to infected nodes small infected areas in the pancreas but did not produce an acute pancreatitis. He, therefore, because of these results and because he cannot believe that lymph would actually flow backward into the pancreas, concludes that "the clinical proof which has always been based on the assumption that experimentally and anatomically, the lymphatic route of infection was possible, must of necessity be regarded as non-existing." But here again we can find no incompatibility with Sweet's theory as outlined above. A localized area of pancreatic necrosis may be caused by contiguity from infected nodes, or by contact with a duodenal or gastric ulcer. A possible etiology by a hematogenous route has been suggested by some authors. Hunt[8] asks why since the parotids are known to be subject to specific infection, the same could not be true of the pancreas.

Acute pancreatitis occurs as a rule in middle-aged individuals, especially in those who are overweight. It is slightly more common in men than in women. Alcoholism and diseases of the biliary system are considered contributory causes.

Correct diagnosis is only occasionally made before operation. This is undoubtedly due to the fact that the disease is comparatively so rare in occurrence. The attack is ushered in by a sudden terrific pain in the epigastrium. According to Moynihan,[9] "of all the pains the human body can suffer, this is by far the worst." It may radiate to the back or either shoulder, especially the left. As a rule it is attended by marked collapse with rapid thready pulse, shallow breathing with characteristic dyspnea and fall in temperature. The temperature will rise on the second or third day. Vomiting generally occurs and is interspersed with retching and hiccough. Jaundice may or may not occur.

Examination of the abdomen shows moderate distention with tenderness and rigidity especially of the epigastrium. Tenderness in the gall bladder region extending beyond the midline is very suggestive. There occasionally is bulging of the flanks and the presence of fluid may be detected. Rarely it is possible to feel a mass in the region of the pancreas. A diagnosis of high intestinal obstruction or perforated ulcer is most apt to be made. In intestinal obstruction the pain is not apt to be as severe nor is collapse as marked. The vomiting occurs with little or no nausea, may be projectile in character and if long continued may become fecal in type. In perforated ulcer, a history of previous ulcer symptoms, obtained in about 50 per cent of cases, is of value. Tenderness and the rigidity which is generally more severe extend over the whole abdomen and are not more marked in the epigastrium than elsewhere. The presence of air under the diaphragm as

shown by x-ray would clinch the diagnosis of a perforated viscus. Lowei's mydriatic test has been of value in some cases. This consists of putting four drops of 1/1000 adrenalin in one eye, repeat in five minutes and examine at the end of thirty minutes. Dilatation of the pupil is a positive test. Glycosuria is not an infrequent finding and if it can be determined to be of recent origin, may be helpful in making the diagnosis.

Treatment consists of early operation. When the abdomen is opened, the presence of blood-stained fluid and areas of fat necrosis makes the diagnosis. However, these latter may be absent or may be confined to the root of the mesentery in the immediate neighborhood where they should be carefully looked for. In the more advanced cases the pancreas is felt to be enlarged with a tense capsule and depending upon the stage of the disease, there may be areas of softening or fluctuation. Operative procedures vary, depending upon the pathology present and the condition of the patient. In the very early cases where there is no pus or fluid present cholecystectomy seems to be sufficient. The general practice at present is to drain the biliary system either by means of a cholecystostomy or if the patient will stand a cholecystectomy by a tube in the common duct. All the fluid in the peritoneal cavity should be removed by suction and a drain placed to the capsule of the pancreas. If there is definite softening of the pancreas or abscess formation, the capsule should be split and a drain placed into the pancreas itself. In the very severe cases this is all that should be done as in these cases drainage is the essential need. Any free necrotic material should be removed, though none should be forcibly torn away due to the great danger of hemorrhage. The drain in the gall bladder or duct should be left in situ for two weeks or longer, but the drain to or into the pancreas can be loosened at the end of three or four days and removed as soon as the discharge diminishes. Bailey[10] suggests the use of an ointment containing 2 per cent of hydrochloric acid to prevent the digestion of skin around the wound by the pancreatic ferment. Errors in carbohydrate metabolism should be carefully watched for and suitably treated. As hepatic insufficiency and a depletion of glycogen reserve are likely to be present, glucose infusions should be given both before and after the operation. Recurrence of the disease may take place after an apparent cure as is illustrated by one of our cases (No. 6).

Following is a summary of twelve cases operated on the First (Cornell) Surgical Division of the New York Hospital since 1914.

1. Male, aged thirty-nine. Dull pain in epigastrium for one week, vomiting for three days. No bowel movement for three days. Moderately alcoholic. Large man, cyanotic, diffuse rigidity and tenderness of abdomen most marked in epigastrium. Temperature 101.6°F; urine, no sugar. Operation, sweet-smelling turbid oily fluid in peritoneum. No fat necrosis. Pancreas enlarged and indurated. Cigarette drain to pancreas. Died sixteen hours postoperative. Autopsy showed multiple abscesses.

2. Male, aged thirty-two. Sharp cramp-like pains in lower abdomen for four days. Nausea but no vomiting. No bowel movement in three days. Obese man, distended abdomen, rigid in right upper quadrant, tender to left of umbilicus. Temperature 102°F. Operation turbid serum especially in lesser sac. Multiple hemorrhagic areas. Pancreas swollen. No definite fat necrosis. Lesser sac and gall bladder drained. Died five hours postoperative.

3. Male, aged forty-five. Pain in abdomen for one month, severe for eighteen hours. Vomited once, moderate belching. Marked rigidity and tenderness on right side. Temperature 100°F. White blood count 1800, 87 per cent. Operation, small amount of bloody fluid, fat necrosis. Pancreas necrotic, hemorrhagic and soft. Drain to pancreas. Left hospital cured on eleventh postoperative day.

4. Male, aged fifty-seven. Pain in epigastrium with vomiting for two days. Indigestion for one year. Obese man, distended abdomen, fluid wave present, tenderness in epigastrium,

markedly cyanotic. Operation, large amount bloody fluid, fat necrosis, pancreas large, swollen, indurated. Temperature 101°F. Died on operating table. Was operated at family's insistence though moribund on admission.

5. Male, aged forty-three. Pain in epigastrium for six days. Well nourished. Abdomen hard and boardlike, tender all over. Temperature 97°F. White blood count 7600, 72 per cent. Operation, oedematous gelantinous infiltration of peritoneum. Pancreas swollen and soft. No fat necrosis. Cholecystostomy. One stone present. Home on thirty-third day.

6. Male, aged forty-one. Epigastric pain for two days. Vomited several times. No bowel movements for two days. Two previous attacks. Moderate distention, tenderness all over, especially right upper quadrant. Temperature 101.6°F. Blood count 14000, 78 per cent. Operation, small amount straw colored fluid. Spots of fat necrosis. Large hemorrhagic mass head of pancreas. Gall bladder normal. Cholecystostomy and drain into mass of pancreas. Discharged on seventeenth postoperative day. Readmitted thirty-seven days after discharge. Had been well for four weeks. Similar symptoms and signs. Operation showed pancreas two and a half times normal size. No fat necrosis. Gall bladder and pancreas again drained. Died first postoperative day.

7. Female, aged fifty-three. Severe pain right upper quadrant for two months; had had it at intervals for eleven years. Obese, tender across center of epigastrium. Temperature 103.6°F. Blood count 17,200, 84 per cent. Operation, moderately distended gall bladder containing stones. Pancreas enlarged throughout. Fat necrosis present. Mass in upper surface of pancreas contained 1 oz. of thick pus. Some bleeding. Pancreas packed. Gall bladder drained. Died third postoperative day.

8. Female, aged twenty-eight. Sharp pain left upper quadrant for eight days. Nausea and vomiting for two days. Mass right side, tenderness, no rigidity. Temperature 100–102°F. Blood count 15,000, 92 per cent. Operation, whole pancreas acutely inflamed, fat necrosis. Gall bladder small, infiltrated. No stones. Gall bladder drained. No drainage of pancreas. Ten weeks later a pancreatic abscess was drained. Died sixteen hours postoperative.

9. Female, aged thirty-seven. Epigastric pain for three days. Vomited. Similar attacks for six years. Obese, tenderness and rigidity in right upper quadrant. Temperature 100–102°F. Blood count 10,500, 78 per cent. Operation, cholecystostomy. Fat necrosis throughout. Hemorrhagic areas in body and tail of pancreas. Reoperated for secondary hemorrhage two days later. Died.

10. Female, aged twenty-six. Operated eighteen months before for acute cholelithiasis. Pain in right upper quadrant and epigastrium for three days. Vomiting. Tenderness and rigidity in right upper quadrant. Temperature 101–102°F. Blood count 17,800, 92 per cent. Operated, stone removed from common duct, pancreas swollen. No fat necrosis. Duct drained. Discharged cured four weeks later.

11. Female, aged sixty. Pain in right upper quadrant, back and right shoulder for four days. Vomited. Similar attack five years before. Distended abdomen, contained fluid. Rigid in upper part. Tender. Temperature 101°F. Blood count 10,400, 84 per cent. Operation, pancreas enormously enlarged, fat necrosis. Gall bladder contained stones. Gall bladder and pancreas drained. Died thirtieth postoperative day. Autopsy showed pancreas to be largely destroyed.

12. Male, aged forty-four. Abdominal cramps, nausea and vomiting for twenty-four hours. Abdomen full and distended, rigid and tender on right side. Temperature 101–102°F. Blood count 15,150, 87 per cent. Operation, pancreas swollen and indurated. Fat necrosis and blood-stained fluid present. Drain placed to pancreas. Discharged cured on seventy-fifth day.

Of 12 cases 7 were males and 5 females. There were 8 deaths, a mortality of 66.66 per cent.

It would seem from these cases that earlier diagnosis and treatment of gall bladder disease might prevent the development of pancreatitis in certain cases.

I desire to express my appreciation to Dr. Charles L. Gibson for permission to publish these cases all of which were operated in his services at The New York Hospital.

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SOLITARY CYST OF THE KIDNEY WITH RESECTION*

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LARGE, single or solitary cysts of the kidney are of sufficient frequency to find reports on one or several cases in medical literature every year. The usual recommendation of treatment in the textbooks and the literature has been: resection of the cyst. However, nephrectomy has been the procedure in the larger proportion of cases reported in spite of recommendation. I have seen 14 of these cases, 7 in the past year, 4 of which were personally operated upon by me. Of all these cases only 1 on the operating table apparently had the possibility of a successful removal. In some instances the cases had cysts so large that the kidney itself was merely a thinned-out capsule of renal tissue, so to speak, attached to the outer surface of a large cyst. In other cases the cyst occupied the center of the kidney, with an upper pole and lower pole of renal tissue so widely separated, and the cyst itself so closely attached to the pelvis in the center, that a resection of the cyst, and a reunion of the two widely separated portions of the kidney seemed very difficult, if not impossible of accomplishment without leaving a permanent renal sinus and the possibility of an ultimate nephrectomy. In these 14 cases all were nephrectomized except one.

The resected case is as follows:

G. O. P., a physician, fifty-four years old, seen on July 22, 1929, complaining of pain in the right side of the abdomen, radiating to his right testis. He had had a similar pain three years previously occurring on his left side, followed by hemeturia. At that time the pain was attributed to a renal or ureteral calculus, but none was passed and none found on *x*-ray. *X*-ray at that time showed very large irregularly shaped kidneys, and a tentative diagnosis of polycystic kidneys was made. As a result he lessened his activities and gained 30 lb. in weight. After a year he became afflicted with dizziness and hypertension. A return to active work and reduction in weight brought his blood pressure down and relieved his dizziness.

When seen the patient was a stout, sturdy man, normal except for some tenderness in his right flank and a large mass, apparently the right kidney. The urine was normal.

Cystoscopic examination on July 24 revealed a normal bladder and urethra. Both ureters were easily catheterized to the kidneys. Clear urine was obtained from both sides, the flow from the left being twice that from the right in the same time. Indigo carmine appeared from the left kidney in three minutes and from the right in three and one-half minutes. In ten minutes the concentration from the left kidney was twice that from the right.

Bilateral pyelograms were done and showed a right kidney high up, with a slightly deformed pelvis and a round smooth mass below, evidently the lower pole of the kidney. The left kidney showed a large upper pole with an irregular cavity in the pole communicating with the pelvis.

Diagnosis was made of a large solitary cyst of the lower pole of the right kidney and a

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smaller cyst of the upper pole of the left kidney ruptured into the pelvis.

Operation was performed on July 25 and a large solitary cyst of the right kidney was removed by resection of the lower half of the kidney. The bleeding was profuse but was controlled by cross mattress sutures. The cut end of the kidney was covered by a thick flat pad of the lower part of the fatty capsule. A cigarette drain was placed outside of the fat and the wound closed. The patient had an uninterrupted recovery, the wound discharging a moderate amount of serosanguineous fluid. He was discharged on the fourteenth day.

The patient was apparently normal until the seventeeth day when following exercise and straining he had pain in his right side. This was followed by a chill and high fever, with passing of cloudy, burning urine.

Patient was readmitted to hospital on August 15 and the wound opened and several ounces of pus evacuated. The wound then drained freely of urine, followed by a recession of temperature. He passed urine containing pus and liquid fat. The fat would collect and harden on top of his urine. Wound was revised on September 1 and again on September 19 when his kidney was examined. At that time the fat pad over the lower renal pole had practically disappeared. The wound then rapidly closed up. With the administration of methylene blue by mouth, the wound coloration ceased in ten days following the last revision.

Cystoscopic examination on October 1 showed that his right kidney secreted a few pus cells, 0.8 gm. of urea per liter and had an indigo carmine appearance time of three minutes and equal with the left output in ten minutes. His left kidney was free from pus and secreted 0.6 gm. urea per liter. Pyelograms were not done at this time because of the risk of reopening the healed renal sinus.

In history this case was seen to fit the average. The cysts usually are silent and only discovered by accident although at times they may give rise to pain. The discovery of these cysts were by examination and *x*-ray. The pyelograms were characteristic. In studying these cases we have brought out that there is a peculiar clubbing and shape to the calyces; that the cyst is usually round and that the outline is clear, sharp, distinct and at times almost capsular in density of outline; that the outline of the kidney tissue is distinct from the cyst, of a different

Fig. 1.

density, and at times can be definitely made out through the cyst. These findings are not those found in tumor.

This case was unusual in that it was the first in a fair number that had the aspect of having a fair chance in resection. Moreover there was a cyst on his other kidney which introduced the additional factor of conservation of the right kidney. In resection the kidney tissue was not cut straight but was beveled into the kidney in the shape of the cyst. This undoubtedly left cut renal tubules that did not communicate with the pelvis after the resection. They must have secreted urine under the fat flap and gradually necrosed it, and have broken down into the pelvis, forming an abscess at the lower pole. The cyst on the left kidney was silent and the kidney apparently normal, which has given rise

to the idea that when next we encounter a non-removable solitary cyst, we open the cyst into the pelvis or calyx through pyelotomy incision and then plicate the cyst. We have tried stripping these cysts out of the kidney in the laboratory, as we strip an ovarian or any other cyst, but have found it exceedingly difficult to do so without tearing the cyst to pieces. In the resection we agree that it is best to cut straight through renal tissue well away from the cyst.

MECHANICAL APPENDICITIS*

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NUMEROUS foreign materials are frequently reported as causing appendicitis. Most often the oxyuris vermiformis, other intestinal parasites, cornflower and grape seeds, cholesterol calculi, shot, pins, etc., are the etiological factors. Fecal concretions are very common but recently I have encountered one of unusual dimensions causing an unusual type of pathology.

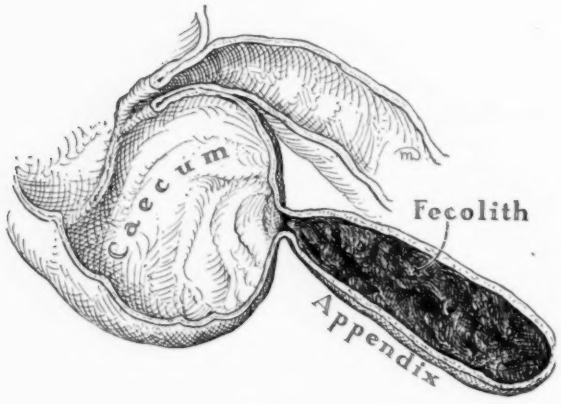

Fig. 1.

Mrs. N. D. gave a history of several attacks of pain in the right lower quadrant over a period of two years. The present attack had been in progress about twenty-four hours when I first saw her. She described the pain as coming in waves, or intermittent in character with no definite time relation between the crest of each sensation. At the height, the pain would make her nauseated, but she did not vomit. Examination revealed a definite tenderness and rigidity of the muscles of the right lower quadrant. Vaginal examination showed nothing more. The pulse and temperature were normal and the white blood cell count 10,500.

A diagnosis of recurrent appendicitis was made and owing to the patient's discomfort, an immediate operation was advised. A right paramedian incision was made. A very large, gray-walled appendix was found. It was the size of a man's thumb and abruptly tapered at the base. It was removed by the usual ligation and purse-string inversion method. The patient made an uneventful recovery and has had no recurrence of the pain since her operation.

The pathological examination revealed that the lumen of the appendix contained one enormous fecalith, measuring 7.4 cm. long, by 2.0 cm. in diameter. The wall of the appendix showed very little change, except for some edema.

Of special interest was the size of the fecalith with such a small degree of associated inflammatory reaction, and also the apparent movements of the fecalith which undoubtedly caused the patient's pain, since it was too large to force its way into the cecum.

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EDITORIALS

POSTOPERATIVE PULMONARY EMBOLISM AND THE REVIVAL OF ARTERIAL EMBOLECTOMY AND THE TRENDELENBURG OPERATION IN SCANDINAVIA AND GERMANY

THE presence in this country of Drs. Gunnar Nystrom, of Upsala and K. H. Giertz, of Stockholm, and the contribution of their personal experiences to the proceedings of The American Surgical Association at its recent meeting in Philadelphia bring to mind the debt of the profession to the Scandinavian surgeons for their initiative and success in the surgical treatment of postoperative pulmonary embolism and the thrombo-embolic obstructions of the peripheral arteries. In fact, the operation of embolectomy for the extraction of obstructing clots from the blood vessels has become a specialty which is a distinctive and almost unique feature of Scandinavian surgery. The numerous scientific contributions of the Swedish surgeons and their unusual experience in the surgical treatment of these thrombo-embolic complications have served to rehabilitate and revive the practice of embolectomy, which up to fifteen years ago gave little promise of the limb and life-saving value which they have since given it.

To those who are at all acquainted with the history of Scandinavian surgery, including Sweden, Denmark, Norway and Finland in this designation, the great impetus given by the surgeons of these northern countries to vascular surgery, particularly in its thrombo-

embolic relations, their activities and their recent achievements are not surprising. It would seem that this is something of a tradition with them which has been handed from the older to the younger generation as a national characteristic, to be cultivated and perpetuated as a splendid inheritance.

It would be impossible to do justice to the long list of Scandinavian surgeons who since the days of the great Danish pathologist Panum, of Copenhagen, have enriched the literature on thrombosis and embolism in all its complex phases by their notable and, in many instances, epochal contributions. Let us not forget that it was Panum who, next to Virchow, in the fifth decade of the last century, developed and expanded the concept of embolism in a series of experimental researches that have remained fundamental and classical to this day.

In later years, Sweden has taken the lead in this specialty. For instance, the collective statistical studies on postoperative pulmonary embolism contributed by G. Petrén of Lund in 1913 constitute an outstanding landmark in the history of this subject. In this masterly essay Petrén surveyed and summarized all the experience that had accumulated on pulmonary embolism as a surgical complication up to the time of its publication. In this he laid the foundation for the differential index of incidence of this formidable complication as it appears after operations on different organs and regions, at different ages and in various diseases, and it is Petrén's work which has served as a statistical model ever since. Of course, the names of Lennander, the great master of Upsala, of Borelius, of Axel Key and Retzius of Bull, of Nicolaysen, of Hansen, of Bauer, of Key and of Fähraeus and Jacobaeus (in cognate lines) and of so many other medical representatives of the great Scandinavian family are familiar to the readers of the *Acta Chirurgica Scandinavica* as eminent contributors to the knowledge and literature of this special field.

It was reserved, however, for the surgeons of the present and rising generation to crown the labors of the pioneers by attaining the highest peak of surgical success as we see exhibited in the achievements of the last few years.

There are two phases of embolectomy that the Swedish surgeons have especially developed. One is the dramatic and heroic operation that Trendelenburg, of Leipzig, devised and first applied in 1908, twenty-two years ago, for the extraction of massive obstructing emboli of the pulmonary artery which, with increasing frequency, contribute to postoperative morbidity and mortality; and the other is the operation of embolectomy for the removal of obturating clots in the peripheral arteries. In both of these procedures the Scandinavian surgeons have set an example that is now serving as a stimulus and encouragement to the surgeons of other countries to repeat their performances. According to Prof. Einar Key, of Stockholm, himself the foremost exponent of the surgery of the peripheral arterial emboli, 216 embolectomies for the removal of clots in the main surgical arteries, including 11 for clots in the abdominal aorta, had been recorded in the world literature up to 1929, and of these, 146 or practically 68 per cent had been performed by the Scandinavian surgeons, Key himself having contributed 15 of these experiences with 8 recoveries, including one successful aortic embolectomy. When we remember that the first embolectomy was performed by Ssbanejew in 1895, thirty-five years ago, and that during the years that followed (to 1911) only 11 embolectomies or thrombectomies, as they were then called, had been recorded in the literature, that up to that time only one patient had recovered (Lahey, Boston), and that the number of recorded operations has since increased to 205, with 146 of these to the credit of the Scandinavian surgeons, we have good reason to believe that this operation has become a specialty in their hands. This fact is emphasized by Pemberton's (Mayo Clinic) statement that up to 1927 only 20 embolectomies had been recorded in the United States, Canada and Great Britain.

The success of the Scandinavian methods is not due solely to their excellent technic, which is practically the same as that laid down by Carrel for arteriorrhaphy, with the free use of sodium citrate solution as an anti-coagulant, but it is in their alertness

in diagnosis and in their promptness to act on the diagnosis that they excel. Their experience has shown that an embolectomy has infinitely better chances for success when performed within the first ten hours and that beyond this time the recurrence of the clot is almost certain and the damage done to the peripheral circulation is irremediable. This is particularly true of the infectious emboli which are thrown out of the heart in septic endocarditis and other infectious diseases in which the embolus is the carrier of a microbial colony that sets up an endarteritis at the seat of embolic obstruction. When the operation is performed within a few hours after the artery has been obstructed, the permanent restoration of the circulation may be expected in as many as 50 per cent of the cases. The graver conditions in which the deobstruation of the arteries is performed in patients suffering from advanced endocarditis and as complications of other systemic infections and cardiovascular diseases, add greatly to the credit of our Scandinavian colleagues for their remarkable success (34 per cent recoveries in 135 operations performed within the first ten hours, Key).

But even more closely related to the postoperative complications that at present engage our attention is the encouragement that we derive from the experience and courageous enterprise of the Scandinavian surgeons in attacking the far more serious and forbidding problem of the acute massive and fatal emboli of the pulmonary artery.

In this performance they share the honors of a great surgical advance with their German colleagues who have persevered in the seemingly cheerless path laid out by Trendelenburg in 1908. Up to 1924, 20 cases of Trendelenburg's operation had been reported in the literature, but it is probable that other unsuccessful attempts had been made which did not find their way to the medical press. All the cases reported up to that time ended fatally during or shortly after the operation. The longest survival was Krüger's patient who lived five and a half days after the operation, when death occurred from a secondary empyema. Since 1924, a very decided change in the prospects of this operation has occurred and now the efforts of the bold pioneers have at last been rewarded by success. Since 1924, 7 successful Trendelenburg operations have been reported, one by Kirschner in 1924, one by Meyer of Berlin in 1927, two by Giertz and Crafoord of Stockholm in 1927, and in the same year another by Nystrom of Upsala. Two other patients were operated on by Nystrom between 1927 and 1928. One of these survived the operation thirty hours, and the other five hours. All these patients were *in extremis* when operated on and therefore the seven recoveries were virtually so many resurrections.

Performance of these operations has taught many valuable lessons; among these is the fact, ascertained by Trendelenburg and since confirmed by more accurate observations, that death does not occur instantly with the onset of the first symptoms as formerly believed. Of 20 cases that came under the observation of Giertz and Crafoord in which the time between the first symptoms of the attack and death was recorded, in only 3 did death occur in less than ten minutes, which is, theoretically at least, sufficient time for a trained surgeon in a well-organized hospital to perform the operation. That no more than 3 patients were actually operated upon is due, as these authors state, mostly to the traditional fear of the operation, the only one that can give hope to save life. In some cases the age, obesity of the patient and other existing complications (pneumonia, cardiovascular diseases, etc.) or previous minor attacks, restrained the surgeons from operating.

Another lesson taught by these experiences is the tolerance of the brain and medulla to a suppression of the circulation and respiration for a much longer period than was at one time deemed possible in

view of previous experimental evidence. Läwen and Sievers had set the time limit of survival after total occlusion of the pulmonary artery to two and a half minutes, which in rabbits could be experimentally prolonged by artificial respiration, oxygen and adrenalin, to seven or eight minutes. Trendelenburg had estimated forty-five seconds as the longest time for survival after a total occlusion of the pulmonary artery in the human subject. In Nystrom's cases the circulation was cut off sixty, sixty-five and, in the patient who recovered permanently, one hundred and four seconds. In Giertz and Crafoord's cases the constrictor was kept on the pulmonary artery for forty-five and sixty-one seconds, the whole procedure taking not more than six minutes (Crafoord).

The operation is usually performed without anesthesia, as the majority of patients become unconscious shortly after the attack. None of the patients showed signs of residual postoperative brain lesions when consciousness returned in from fifteen minutes to six hours after the operations, despite the prolonged suspension of the circulation and respiration. A successful Trendelenburg operation leaves no secondary cardiac or pulmonary lesions and the patients after recovery have remained in perfect health for years (Kirschner's patient over four years).

The operation seems to have gained full acceptance in Europe, particularly in Sweden and Germany where it is a regularly standardized procedure.

The operation has been much improved by Prof. A. W. Meyer, of Berlin, who recently visited the United States. He insisted on the importance of avoiding the pleura to prevent the collapse of the lungs from pneumothorax, a particularly serious complication in these circumstances and one which undoubtedly contributed to the mortality following the earlier operations performed by the original Trendelenburg method. He also allowed the pulmonary circulation to be reestablished while operating by resorting to intermittent partial occlusion of the pulmonary artery. He modified the Trendelenburg instruments, and strikingly demonstrated the great value of adrenalin injections into the aorta and of the inhalations of CO_2 and oxygen mixture as a powerful respiratory stimulant at the critical moment when the respiration is arrested. A suction apparatus, which need not be of the Trendelenburg model, is essential and is of incalculable value in the extraction of the clot. The instruments required are in reality very simple and can be found or improvised in any well-equipped hospital. However, a Trendelenburg outfit modified by Meyer is desirable but not essential for the purpose. What is more important is that the surgeon who may be called to perform the operation should familiarize himself with the anatomy and the technic by repeated exercises on the cadaver and in the experimental laboratory. With this proviso, there is no reason why a well-trained surgeon should not perform the operation. It offers no greater technical difficulties than a cardiorrhaphy for a wounded heart or for the extraction of a bullet lodged in its chambers.

The Trendelenburg operation seems to be going through the historical evolution of all the great epochal operations. There is first the period of scepticism and doubt, then sporadic, tentative and often unsuccessful trials, and finally success and complete acceptance.

The life-saving limitations of the Trendelenburg procedure are obvious. At one time it was thought that one-half of the patients died within the first three to five minutes of the attack. More accurate observation has shown that the majority, on the basis of ten minutes' survival after the onset of the attack, could be operated upon; 20 out of 27 cases were regarded in Giertz and Crafoord's experience as operable under the most favorable hospital conditions. This, however, we must regard as a very optimistic view which may be theoretically correct but practically diffi-

cult, if not impossible to realize, even in the best hospital surroundings. The differential diagnosis at times may offer serious difficulties which justify hesitation and delay. In doubtful cases, Nystrom's suggestion to do a preliminary costochondral resection as preparatory to a later emergency operation and to do this under local anesthesia may be a helpful suggestion in the doubtful cases. However, it is significant that in 23 out of 27 cases that were under the observation of Giertz and Crafoord the clinical preoperative diagnosis was proved to be correct by either operation or autopsy. The worst prognostic feature is the possible recurrence of the embolus or the presence of multiple emboli broken up and lodged in inaccessible branches of the artery. But the 7 patients who have been saved within the last few years prove that the obstacles are not so great that many otherwise doomed patients cannot be saved and permanently restored to health and usefulness. There is no question that much judgment is required in the selection of cases. Nystrom has suggested that in patients suffering from other grave complicating diseases (cancer, tuberculosis, septicemia, etc.) and in the very aged and infirm, the operation would probably only serve as a *coup de grace*.

The visits of Drs. Nystrom and Giertz, preceded a few weeks ago by that of Dr. Meyer, and their discussions of pulmonary embolism and its surgical treatment will no doubt help, in view of their great authority, to kindle a new interest in the Trendelenburg operation. This operation, though magnificently conceived in the fertile mind of its originator, had been followed by such discouraging results that, notwithstanding the early advocacy by Willy Meyer of New York and Matas of New Orleans, it was regarded in this country more as a matter of theoretical interest than of practical application. The recent achievements of the German and Scandinavian surgeons have brought the operation to the attention of the profession in a new light as a practical life-saving measure and have given it recognition as one of the most legitimate triumphs of modern surgery. It is hoped that the teachers in charge of the schools of operative and experimental surgery will take heed of this notable advance and make the practice of embolectomy and of the Trendelenburg operation an obligate part of the curriculum in the laboratories of operative surgery. R. Matas.

MEDICO-LEGAL PROBLEMS*

WHENEVER someone feels inspired and impelled to launch an attack upon the administration of the law, the medical witness is quite apt to be one of his targets. The usual response of the medical profession is the charge that the situation is due to the laws, the lawyers and the judges and usually couples this with a suggestion that the laws and court procedure should be changed. While it is true that the laws and court procedure can be improved upon and that judges and lawyers could bring about a more efficient administration of the law even without any changes being made in the laws, it is also true that the attitude of the public toward the testimony of medical experts is largely chargeable to the medical profession itself. I refer particularly to the major criticisms that expert witnesses differ so radically in their opinions and that their testimony is so often unsatisfactory and unconvincing.

Differences of opinion between expert witnesses, when analyzed, are traceable to perfectly natural causes. Where two men, though technically qualifying under the law as medical expert witnesses, differ

* An Address Delivered to the Los Angeles County Medical Association, February 20, 1930.

substantially in their qualifications of knowledge and experience, a difference of opinion may naturally be expected. Such a situation, properly handled by the court and counsel, will clearly disclose the proper weight to be given to the testimony of the respective witnesses. Improperly handled, the witnesses may appear to be equally qualified and the appearance of unjustifiable conflict of opinion thus created may be charged to causes not at all to the credit of one or both of the witnesses. In every phase of a court trial the skill of the lawyer, particularly that of the lawyer against whom an expert witness is produced, is a big factor in the weight which will be given to the testimony and this is especially true where the opposing counsel is distinctly less qualified. But a trial should not be determined by the skill of counsel. The judge should be more than a mere umpire ruling upon the objections of counsel. It should be his function to assist counsel in securing a fair presentation of the facts. He should also protect witnesses from unfair cross-examination and discourtesy of counsel and, when it appears that the counsel producing a witness has not placed his testimony fairly before the court, the court may and should ask of the witness those questions which will bring out the proof which the witness can produce. If a witness, posing as an expert, does not appear to be well qualified, it is not only proper but a true function of the court to inquire into his qualifications and the reliability and accuracy of his testimony.

As between experts of comparatively equal knowledge and experience a difference of opinion may be either honest or dishonest. It is a pleasure to say sincerely that in a trial experience of over a quarter of a century as a trial lawyer and a judge I have encountered but one medical witness who was not honest in the testimony he gave and he was thoroughly discredited at the trial. We may therefore dismiss the dishonest expert as a wholly negligible factor. Differences of opinion between honest, qualified experts may well arise just as do honest differences of opinion between any two persons over the same set of facts. Particularly is this true in forensic medicine since it is rarely the case that the experts have at their disposal all of the material facts. Most differences of opinion between experts may be traced to a difference in the facts upon which the opinions are founded. Naturally each side of a lawsuit has a theory upon which it bases its contentions. Quite naturally the facts in the possession of the plaintiff include matters not known to the defense and information possessed by the defendant is often not known to the plaintiff. Furthermore there is a natural tendency for each party to minimize the facts which tend to support the theory of the opposite party and to lay too great a strength with the facts in his own favor. This tendency is quite prominently indicated by the statements of fact as furnished to the partisan expert. There is also a tendency to withold from the expert those matters of information which are disputed or disbelieved. It quite naturally follows that the experts for the plaintiff receive a different statement of the facts than that which is received by the experts for the defense. Furthermore there is not always allowed to the experts for the adverse party the same freedom and latitude of examination which is afforded by the party to be examined to his own experts. To these factors must always be added the element that the experts hired by a party to diagnose his condition are more prone to believe his statements as to his condition and symptoms and the adverse experts are more likely to carry a suspicion of possible malingering. These elements and some others of more occasional character are inherent in the system of trial where each party provides his own experts. As suggesting a remedy or improvement let us consider for a moment as a concrete topic the insanity defense in a criminal prosecution.

Before the amendments to the California statutes relating to the defense of insanity, it was the usual thing for each side to have its own experts; the prosecution produced experts who were of the opinion that the accused was sane while those produced for the defense were of the contrary opinion. I have a suspicion that in some cases one of the parties failed to call as a witness an expert who, though originally employed, did not hold an opinion consistent with the theory of the party who sought his services. Under this form of procedure the facilities afforded the prosecution alienists were ordinarily not the same as those afforded the doctors for the defense. It was not at all an unusual occurrence to find defense counsel insisting on being present when the prosecution alienists examined his client, frequently, when present, interfering with and preventing a proper examination. In one instance an attorney for a defendant absolutely refused to permit an examination of his client by the doctors for the prosecution and did not consent to such an examination until after the trial had actually commenced and then only when informed by the prosecutor that, if he persisted in his refusal, the fact that he had prevented such an examination would be proved at the trial. Another factor which cannot be ignored when experts are employed by the respective parties is the influence, conscious or subconscious, which is likely to result whenever an expert is employed, especially when his employment comes from a lawyer of high standing in his profession and in whose honesty and sincerity the physician has full confidence. Quite naturally under the old system of procedure the trial, so far as the issue of insanity was concerned, became a contest of experts and lawyers and, in the heat of that contest it was to be expected that the experts would develop not only a tendency to become partisans but at times even became champions for their respective sides of the question. After all it is rather human to enjoy a good scrap.

Under the present law of California, where the court, immediately upon the entering of a plea of "not guilty by reason of insanity," appoints a committee of experts to investigate and inquire into the merits of the defense of insanity and to render a report to the court of their findings, it has been the experience that the experts appointed substantially agree in their conclusions and, where differences of opinion exist, they are reasonable differences of opinion,due to the manner in which the symptoms, history and other factors were viewed. While the present law has been in operation only since August 1929, it has been invoked with sufficient frequency to justify the belief that it will operate as well in the future as it has in the past and its success and practicability are chiefly due to the following factors: First, the experts are not selected by, or in any sense bound to or compensated by, either the prosecution or the defense. Second, the court in making its appointments is entirely free from any partisan desires in the selection of the experts but selects its commission from among those members of the medical profession whom it deems well qualified. Third, and perhaps most important, the experts are each given a full and equal opportunity of learning all about the case unhampered by the interference of counsel. Of especial importance to the medical profession is the fact that, since the experts are appointed by the court and hence are non-partisan, their testimony is more nearly accorded the weight to which it is entitled, for the jurors look upon the experts appointed by the court as somewhat in the nature of officers of the court and, as such, entirely free from any partisanship or interest in the ultimate outcome of the case. Under this new order of things also, the attitude of the attorneys has undergone a distinct change. Cross-examination has become more of an inquiry to elicit information in addition to that which was brought out on direct examination, rather than, as was the former usual practice, an attack directed against

the witness. Trial lawyers of ability are fully aware of the prejudicial effect of any effort on their part to attack or discredit an expert witness appointed by the court, virtually vouched for by the court. While both the prosecution and the defense retain their former right to bring in experts of their own choosing, in addition to those appointed by the court, it is apparent from the past experience that this production of partisan experts is most unlikely to occur. If partisan experts should be called in and a conflict in testimony should arise between them and the experts appointed by the court, all natural tendencies would cause the jurors to give greater weight to the testimony of the court appointed experts. It is believed by those who have closely followed the operation of the new laws relating to the insanity defense that they come closer to producing a just verdict or decision than was possible under the former procedure. A method of procedure which works so well in cases involving questions of psychiatry is certainly worth trying in all cases involving expert testimony. Even under the new law relating to the defense of insanity there is much room for improvement, particularly in the provisions relating to the selection of experts by the court. That provision which arbitrarily requires that at least one of the experts appointed must be connected with one of the state hospitals is particularly impractical in those counties far removed from any one of the state hospitals and it is always unwise to interfere with the full exercise of the discretion of a court, particularly in the matter of the designation of experts in whom the court has personal confidence. The law is also somewhat ill advised in that it requires the commission of experts to consist of two or three members. While the courts no doubt have the power to nominate more than three experts, it is mandatory that at least two be appointed. It is well known that there are cases in which the insanity of the defendant is so obvious that no good reason can exist for the cumbersome and more expensive method which requires more than one expert.

Much as we may deplore it, the fact remains that the general public has a lack of confidence in medical testimony and that this sentiment exists in the minds of many jurors and, to an extent at least, in the minds of the judges. The fact that this impression is largely unwarranted does not alter the unfortunate situation. But, since it does exist, it must be met; as far as it is unwarranted it must be overcome, and, insofar as it is justified, an effort should be made to remove the cause. Until this is done, every medical expert called as a witness must not only give his testimony but must also overcome any prejudice which may exist in the minds of those members of the general public who have to pass upon his testimony. This handicap of prejudice is due in no small part to the errors of commission or omission of the expert medical witnesses who have gone before him and is not an inherent characteristic. In foreign countries medical testimony is given far greater consideration than it is in this country and the expert is looked upon by the court and jury as a man whose testimony will bring to their assistance the full benefits of modern scientific knowledge.

While the gaining of the confidence of the court and jury depends in no small measure upon the personality of the witness, his conduct and appearance upon the witness stand; the weight which will ultimately be given to his testimony depends most largely upon the impression which the testimony itself will create. The giving of expert testimony in court is an art which is not inherited but must be acquired. No physician can say with any assurance that he will not be called into court during the coming year to render some medico-legal service and still, unless he be one of the rare few who, by study or consideration or actual experience or their combination, have learned the art of giving expert testimony,

the chances are that, if he is called, he will not only fail to do himself justice but will not render that degree and quality of service which modern medical science can bring to the assistance of the court. While this must naturally follow where the doctor is not possessed of a sufficiently thorough and accurate knowledge of the subject involved, a similar result follows where, even though the physician possesses the necessary skill and knowledge of the medical question involved, he lacks a knowledge of the manner of solving the question referred to him both as to the manner of conducting his investigation and as to the manner of testifying after he arrives in the courtroom.

The skillful trial lawyer is always mindful of the fact that a jury is composed of average citizens of the community to whom the matters and subjects involved in the trial are largely new and novel. Consequently he directs his efforts not only to the end of proving the facts making up his case but to proving them in so clear and conclusive a manner that every juror not only understands every word of the testimony but is convinced thereby. More than half of the verdicts of juries which have been criticised as being an unjust determination of the issues involved are more properly chargeable to the manner in which the lawyers and witnesses performed their functions in the courtroom. Both the witnesses and counsel must constantly take into consideration that the jurors are just average citizens, that their duties as jurors are most likely different from any experience they have had theretofore, that many of the facts in the case involve matters of knowledge which are entirely new to at least a portion of the jury and that the witness is rather an educator than merely an individual stating ultimate facts. It is not at all unusual, when discussing with a juror the facts of some case in which he served, to find that he had arrived at a conclusion on the facts not warranted by the testimony as that testimony would be analysed by a trained and experienced judge or lawyer. A careful examination and review of the testimony will usually disclose that the real fault lay, not with the juror, but with the manner in which the proof was introduced. The average witness is so familiar with the subject matter of his testimony that he wholly overlooks that he is relating something which is wholly new to his hearers and fails to pay any consideration to the fact that the very purpose of his being on the witness stand is to transfer his knowledge to the minds of the jury. Thus a physician and surgeon who has examined the person of a litigant for the purpose of testifying to his physical condition is quite likely to come into court and testify to his findings in much the same manner that he would report the case to a group of doctors. To aggravate the situation the lawyers, desiring to display their knowledge of medical science (quite often acquired by a brief period of reading of an obsolete textbook) conduct the examination of the doctor in much the same tune, with the results that the jury, unable to grasp what it is all about, directs its attention elsewhere and to something more interesting. This is due in part to the perfectly natural use of scientific terms by the expert. While not a part, except indirectly, of my topic, I should like to repeat what was said on a former occasion that every witness, be he expert or layman, should give his testimony in such language that it will be fully understood by every member of the jury. This must be particularly difficult for the expert witness for, in spite of all that has been said on the question, our experts still testify far over the heads of the jury. Where the proceeding is a trial or hearing before a judge without a jury it may be a delicate compliment to the court to use as technical language as possible, but the decision will be most likely to go to that side whose understandable testimony is most convincing. The ability to testify in non-technical language is and can only result from a

clear grasp and understanding of the subject coupled with careful thought and consideration before taking the witness stand. The expert must develop an ability analagous to that of the successful teacher or instructor.

The point, however, to which I desire to call particular attention here is that of the preparation of the subject matter of the testimony to be given. The expert must always bear in mind that the objective is not that he convince himself as to the ultimate fact or facts but that his efforts must be extended far beyond that point and must be directed to the production of proof so that the final judgment of the court in which the trial is had, shall be in accord with the facts, a result which can only be attained if the judge, or the jury if it be a jury trial, receives all of the proof necessary to such a determination. No judge or jury will accept the opinion of an expert witness merely because he declares it from the witness stand. The opinion should always be supported by the facts upon which that opinion is based. It must be anticipated that, before the expert leaves the witness stand, he will be called upon to give the reasons and basis for his opinion. It would surprise the medical profession if they knew how many of their brother physicians have failed to convince the court or jury because of the incomplete or unsatisfactory basis of their expressed opinions. Take a case in which the doctor has testified that, in his opinion, a defendant is suffering from a particular disease. When called upon to state the basis of his opinion it develops that his opinion is absolutely dependent upon the statements of the person examined and that the doctor's conclusion can, therefore, be correct only if the statements of the patient were true. This reduces the decision of the question of fact largely to the truthfulness of the patient's declarations to the doctor and, the defendant being a very much interested party to the action, it is but natural that the weight given the doctor's opinion is ultimately determined by the credibility of the patient. This type of case occurs quite frequently. Not so long ago I received an opinion from a physician that a defendant who had been convicted of shoplifting, after about ten years of activity in that line of business, was suffering from kleptomania, his opinion being based entirely on the statements of the defendant that she didn't know why she had stolen things and had only stolen articles of small value. This same defendant had made statements after her arrest and had testified at her trial entirely at variance with her statements to the doctor; in other words his opinion was based on only part of the available information. I am satisfied that, had he heard the woman's testimony on the witness stand, he never would have formed the opinion that she was a kleptomaniac. In his private practice the physician is warranted in believing the statements made to him by the patients who come to him for relief. They are not viewed as possible malingerers but are credited with sincerity even though it should ultimately develop that their ills are largely or wholly imaginary. But, when the patient is a party to a lawsuit or criminal prosecution, every effort should be made to guard against being imposed upon by a statement of facts and symptoms which have no basis in fact. Some years ago one of our well-known psychiatrists was appointed by the court to examine a prisoner in jail and later took the stand and testified that, in his opinion the prisoner was insane. Shortly thereafter the same prisoner came into court and pleaded guilty to two robberies, admitted that he had not had any of the symptoms upon which the doctor had based his opinion and that, so far as the objective symptoms were concerned, they had been feigned and then laughed over the way he had fooled one of the doctors. I must admit that this prisoner was one of the most clever malingerers I have ever met and his simulation of insanity was nearly perfect. The fraud was exposed only by

going beyond the prisoner's own statements and appearance at the time of the examination. The interested party in pending litigation may be an out-and-out faker or merely an unconscious malingerer; but such possibilities always exist and must be guarded against. The legal profession is well aware of the fact that the client who will give a fair, accurate and unbiased statement of the facts is an extremely rare individual. It seems almost inevitable that a client, like a young man courting a girl, will put forth in somewhat magnified form everything in his favor and entirely overlook or at least minimize everything to the contrary. This desire to exaggerate is well illustrated by a recent occurrence in a case in which a defendant desired to be released on bail pending an appeal after conviction of a felony. To support his claim that confinement in jail was imminently dangerous to his life, affidavits of physicians were filed in one of which appeared the statement that every meal taken by the prisoner after his commitment to jail several months previous had been forced. The testimony taken upon the hearing showed that there had not been a single instance of forced feeding. In the same set of affidavits it appeared that the statements as to the prisoner's weight as given by him were changed after the affidavits had been signed because it was discovered before the affidavits were filed that such statements of weight were not correct. To an ordinary person it would appear that the accurate way to get a man's weight is to weigh him and not accept his statement where weight is a factor in a diagnosis. What has been suggested in relation to the statements of the interested party himself applies largely to statements of friends and relatives. It is surprising how many conclusively insane acts of a defendant are discovered after he has murdered someone. Statements as to family history and past history of the patient, even in private practice, are very likely to be inaccurate or unreliable. People really know very little about the cause of death of an ancestor or relative; what information they may once have had may be forgotten or, if not forgotten, may originally have been erroneous. This latter situation is quite likely to exist where the actual cause of death or a contributing cause of death is one of those diseases which people prefer not to mention. In addition to these possible defects in the statements of friends and relatives of a party to litigation the doctor must also guard against direct falsehood uttered to assist the litigant. One would hardly expect that the ordinary person charged with a crime could possibly feign insanity and still we have had instances right here in Los Angeles of prisoners who have so successfully feigned the symptoms and appearance of the insane as to fool able psychiatrists. In making this statement I have no reference to those cases in which the only proof of the absence of insanity is the verdict of a jury, but have in mind those cases in which the fact of malingering was conclusively established. If a man of the criminal type can so effectively simulate insanity with its complexity of symptoms, the possibility of a party to a lawsuit feigning a simpler ailment is apparent. The medical expert owes it not only to the courts and the interests of justice to use every possible precaution against deception but is obligated to the members of his own profession to do so lest, should he be deceived into giving an opinion not warranted by the actual facts, he cast discredit upon expert medical testimony in general. The attitude of the public toward the medical expert which now exists is based upon what it has heard and observed of the conduct and testimony of medical experts in the past. This attitude is largely unjustified, especially in so far as it is based upon the facts that experts have given diametrically opposed opinions and, in so far as it is justified it should be limited to those members of the profession who are no credit to it. The remarks in this discussion are largely directed in an effort to point out a means

of eliminating much of this adverse criticism in the future. Every time a physician and surgeon renders medico-legal service it should be of so high a character that it will be a credit to medical science. This means careful study, research and preparation, and a high sense of loyalty and devotion to his profession.

One of the minor contacts between the doctor and the court arises when a patient requests his physician to provide him with a very necessary certificate of disability to explain or excuse his attendance in court, the patient usually expecting a certificate containing as much evidence of trauma or pathology as possible. Having seen a goodly number of certificates of this character I am firmly convinced that the medical profession is not only extremely ignorant of the legal requirements but also rarely, if ever, considers the impression which the certificate will leave with the judge to whom it is ultimately presented. We are all aware and appreciative of the psychological effect of a well written letter on good stationery and, if we gave the matter a moment's thought, we should realize the effect of a hastily scrawled certificate written on a prescription blank. To make matters even worse the actual information which such a certificate should contain, the facts about the patient, are often conspicuous by their absence and the certificate consists usually of no more than an expression of the doctor's opinion as to how long the patient will be incapacitated. Add to all this the chances that the doctor is not known to the judge and it doesn't require higher mathematics to figure out the weight which the certificate will carry. Again, it not infrequently occurs that the certificate unjustifiably represents a condition which does not in fact exist. This is not due to any desire on the part of the physician to mislead the court but because he too carelessly accepts the statements of the patient and predicates his certificate upon those statements. In other instances a physician is called in for virtually the sole purpose of securing a medical certificate from him. Not so long ago a defendant in a criminal case failed to appear in court when required and his attorney presented a medical certificate which his client had sent him. The certificate stated that the patient was suffering from nephritis and prostatitis, was confined to his bed and could not possibly be in court for at least several weeks. The district attorney knew the defendant better than did the doctor and sent two police officers to the defendant's residence to check up on him. They found the defendant up and about without any signs of any disability and on his desk found a letter to a pal, just completed, telling him how he had fooled a doctor into giving him a medical certificate and thereby evaded going to court. The defendant was taken to jail on a bench warrant and, though thereafter kept in jail to insure his presence when required, displayed no marked symptoms of any incapacity. An examination of the physician disclosed the fact that he had seen the patient on but one occasion and that his certificate had been based on what the patient had told him. It does not require many such experiences before a judge looks upon a medical certificate with considerable suspicion unless he happens to know the physician. The only safe course is never to certify to anything that would not be stated in the same manner under oath. Every such certificate should give an accurate picture of the case, stated in plain language, and, when the certificate contains a statement of the probable duration of the incapacity, the reasons for such opinion should be stated. This will entail special effort but then the impression created by a certificate is normally proportionate to the effort displayed. It must be borne in mind in this connection that proof in any court proceeding must be under oath and that the acceptance of an unverified certificate is a matter of courtesy on the part of the court.

In all matters of any particular importance the medical certificate should be in the form of an affidavit and sworn to before a notary or other officer authorized to administer oaths. The affidavit should contain a statement of the doctor's special qualifications as that will largely determine the weight which will be given to his statements and should conform to the suggestions elsewhere made in connection with the subject of oral testimony. It is the common practice for the lawyer to draft the affidavit for the doctor, but if this be done, it should be carefully edited by the affiant. The better practice would be for the doctor to personally prepare the body of the affidavit.

There is one class of cases in which the doctor is called upon to treat or examine a patient or to conduct a post-mortem examination and which eventually become the subject of a trial in court. While the probabilities of the resulting trial can usually be anticipated from the nature of the case it is the rule rather than the exception that the doctor makes no special preparation for his eventual appearance in court. When he does arrive in court we frequently find that his memory is extremely hazy as to the conditions and facts of the case and that his personal records are so incomplete that they are useless as a memory refresher. The law makes it a misdemeanor for any physician and surgeon to fail to immediately report to the local police or to the sheriff if the case be outside municipal boundaries, every case handled by him in which the patient is suffering from an injury caused by a deadly weapon or by a violation of any penal law of this state. This report must state the nature and character of the injuries but, in spite of the obvious wisdom of the requirement, we find only a limited compliance with its provisions, the average report being most meager in its statements of fact. Even independent of the law the minor as well as the major injuries should be carefully noted, not because of their medical but because of their medico-legal and evidentiary value. A few minor scratches on the forehead of a man arrested for driving an automobile while under the influence of intoxicating liquor may be of no interest to the police surgeon who is giving him a test for sobriety; but when the doctor arrives in court he is apt to find himself being asked whether he noticed the injuries to the defendant's head and usually his testimony is that he is unable to state whether any such injuries were present. By the time the trial ends we find the defendant contending that his unsteady condition was due to head injuries received in the collision, a contention which the doctor would have been able to disprove or verify had he performed his full duty. As a police surgeon he should have anticipated that very common line of defense and be prepared to give all the ascertainable facts when called for, not only because of their evidentiary value, but because the admission that he does not know whether the defendant had any head injuries tends to show that his examination was not thorough and his evidence is consequently discounted. In a case of the character last referred to and recently tried, the police surgeon testified that he had no knowledge that the defendant had suffered any injuries when the fact was that he had suffered a fracture of several ribs. In examinations for sobriety the element of thickness of speech is a distinct factor and the defendant quite often endeavors to account for the thickness of speech by claiming that his speech is normally thick and indistinct, that his nationality gives him such type of speech, that he was dazed by the accident or that he had a new set of false teeth. I am still awaiting the testimony of a physician in such a case, differentiating the speech of an alcoholic from defects of speech from other causes.

The location, direction, size and appearance of wounds are of distinct importance in the prosecution of the assailant,

but the private practitioner usually has only the most indefinite recollection as to these matters. Recently a doctor testified in one of our courts to treating a boy who had been shot through the abdomen but was unable to tell whether the wound was on the right or the left side. Naturally juries will discount the testimony of a doctor who is unable to give the information which he should be able to furnish if his examination were thorough, especially as to so obvious and important a fact as the location of the bullet wound. Even in cases of post-mortem examination we find at times a lack of scientific precision. An autoptical examination such as would be made by a first-year medical student is not up to the standard expected of a graduate physician. Some years ago two of our local doctors were present and participated in the post-mortem examination of a man who had been shot to death. During the trial for homicide which followed it became important, as tending to prove the positions of the deceased and the accused at the time the shot was fired, to prove the exact location of the entrance and exit wounds. Both doctors testified positively as to the exact location of the bullet holes, but one of them placed the exit wound six inches higher up on the back than did the other. The district attorney had to exhume the body to settle the question. In another case the lip of the deceased was found cut entirely through. The nature of the instrument causing this particular injury was very important but the autopsy surgeon was unable to express an opinion as to whether the wound had been caused by the sharp edge of a knife or by a blunt instrument like the butt of the handle of a butcher knife. In still another case the autopsy surgeon testified that he had performed an autopsy and that death was due to a gunshot wound of the chest passing through the heart but admitted on further questioning that he did not know what part of the heart the bullet had passed through. Instances of this kind of testimony, and others might be cited, are and have been an important factor in the formation of the regard in which the public holds the medical expert witness.

Much of the cause for the failure of the doctor to be able to give all the details which a thorough examination would have disclosed is the fact that he stopped as soon as he had performed his functions as a physician or had ascertained the cause of death and overlooked his function as a medico-legal expert. Whenever a physician has any reason to believe that he may later be called into court as a witness, his examination and observations should be conducted with scientific accuracy not only as to the principal subject of his attention as a physician, the condition requiring medical or surgical attention, but to every physical sign which could by any possibility be of evidentiary value even though it be a mere scratch. And, since human memory is fallible, these facts should be carefully and fully recorded in a notebook, supplementing the written word by a diagram whenever possible. Such a record is not only valuable for the purpose of brushing up the memory before going into court but the law permits the doctor to consult such a memorandum while on the witness stand for the purpose of refreshing his memory.

When a physician is called in as a medico-legal examiner to examine an individual for the purpose of establishing a fact by the application of the resources of medical science, he should bring to bear upon the problem the highest degree of skill, knowledge and accuracy of detail of which he is capable. This one subdivision of our subject is itself so extensive as to merit a lengthy presentation and citation of illustrations, but this occasion permits of but a single illustration. Let us consider a charge of rape. While there are not many cases of rape accomplished by means of force or fear, most states punish what is known as statutory rape in which the lack of consent of the female is not an

element of the crime, the offense consisting in the commission of the sexual act with a female under a certain age, eighteen in California. In either class of case one of the important facts to be proved is that an act of sexual intercourse took place and in nearly every case the prosecutrix is turned over to a physician for examination for the purpose of obtaining medical testimony on this issue. If an experience of about ten years in the office of district attorney supplemented by about two and a half years as judge presiding over criminal trials were my sole source of information, I would be forced to conclude that all that medical science can do towards determining whether a female has had an act of sexual intercourse, unless pregnancy or venereal disease is present, is to determine whether the hymen has been ruptured and whether the rupture is recent. Even here the doctor must admit that a ruptured hymen may result from causes other than sexual intercourse. During this twelve or more years of experience there were only two cases in which spots on the clothing were examined to determine whether spermatozoa were present; but even a positive finding from such an examination would prove only that there had been an emission and would not prove a penetration of the female sexual organs. It would seem logical that, since in completed sexual intercourse millions of spermatozoa are introduced into the vaginal tract, medical science could discover the presence of a few of them if an examination were made within a reasonable time after the sexual act and that their absence upon an examination shortly after an alleged act would have the tendency of rather conclusively proving that such an act had not taken place provided that the absence of spermatozoa was not accounted for by some additional factor. The answer as to why such examinations are not made would be interesting. So also would be an explanation of the testimony of three doctors in a recent case. Each testified to having examined the prosecutrix; one testified to finding the hymen ruptured, a second that the hymen had not been ruptured and the third testified that he could not determine whether the hymen had been ruptured or not.

Whenever a doctor starts out to discuss the subject of expert medical testimony we may confidently expect that before he concludes he will have criticised court procedure and the rules of evidence. Some of this criticism may be justified but most of it is due to a lack of knowledge or misunderstanding of the law. Criticism of the law by a doctor who is not familiar with its provision may satisfy his personal feelings but gets us nowhere. It is interesting to note that most of the criticism comes from the doctor whose knowledge of the law is based almost entirely upon personal experience in the courtroom. It is undoubtedly often true that the expert medical witness leaves the witness stand with a feeling that he was not permitted to give the testimony he had expected to give. This may have been due to the fact that he did not know how to testify but is more likely attributable to the fact that the lawyer who called him as a witness did not know how to bring out his testimony. The percentage of lawyers who do not know how to put on expert medical testimony is fully as high as that of the doctors who do not know how to testify. Dr. Edward Huntington Williams has given us an excellent contribution on this subject in his recent book, "The Doctor in Court." Dr. Paul E. Bowers, also a member of your association and one who has studied law, at a joint meeting of the Los Angeles Bar Association and the Los Angeles County Medical Association in November 1928, also discussed the expert witness in an able but all too brief address which was later published,[1] and, in concluding, suggested that the two learned professions make a real effort to cooperate in a solution of the problem. Without a mutual understanding of the

[1] *Inter. Clin.*, s. 39, vol. 3.

respective viewpoints of the two professions of law and medicine the problems cannot be solved, for many of the problems are the direct results of misunderstandings. Meetings attended by members of both professions at which the subject is discussed will do much toward improving conditions, but the discussions must be frank as well as friendly even though at times the frankness may not be altogether pleasant. Above all there must be an individual willingness and desire to study the problems and an individual effort on the part of every doctor to make the medico-legal expert just what the term implies.

Charles W. Fricke, J.D., LL.M.
Judge of the Superior
Court of Los Angeles
County, Calif.

FLUOROSCOPIC CONTROL OF FRACTURE SURGERY

During the last decade surgeons have developed a commendable increase in interest in the radioscopic control of their traumatic work, especially during the actual setting of fractures. In a few large hospitals where there is much of this work to be done, the *x*-ray department has been equipped with a special room and a special table for this type of fluoroscopy permitting simultaneous observation in two planes, always depending upon trained medical personnel from the *x*-ray department to do the fluoroscopic work. The example set in these large, well-equipped hospitals has been contagious, and very naturally there has resulted a commendable desire on the part of surgeons generally to utilize the fluoroscopic control in their fracture work. Unfortunately, the majority of hospitals, especially the smaller ones, do not have the full-time services of a physician radiologist, so that many surgeons either by choice or by force of circumstances have come to do their own fluoroscopic observations, usually with a radiological technician turning the current on and off at the command of the observer.

The advance in traumatic surgery represented by this screen control of the setting of the fractures is not without its dangers, however, especially in those cases where the surgeon prefers or is obliged to do his own fluoroscopic observations. Radiological technicians are not, and by the very nature of things cannot be, expert in radioscopy. They are not physicians and at no time in their work or training do they have opportunity or need of equipping themselves to do fluoroscopy. Hence, all that the technician can do in connection with fluoroscopy of fractures is to start and stop the current at the behest of the surgeon. Very few physicians not specializing in radiology have any definite idea of how much *x*-radiation can be safely administered at one sitting; indeed, few have any definite conception of how much radiation is being received by the part per minute. The chief advantage of the footswitch, with simultaneous control of *x*-ray and overhead light, as introduced by the writer into fluoroscopic work in 1908, lies in one's ability to keep the current off at all moments the eyes are not intently fixed on the screen and ready of immediate observation. The current should never be on one single second uselessly, that is, when the observer is not alert and ready to look. This means that the observer will make no attempt to fluoroscope the patient until he has been in darkness or very obscure light for at least fifteen minutes; for any attempt to use the screen sooner will require a much higher milliamperage than is necessary or safe. With a footswitch of this sort adequately used the actual dose of *x*-rays received by the patient may be reduced threefold or fourfold. A sheet of aluminum of 1 mm. thick-

ness should be fixed in place between the tube and the patient for all screen work. In fact, it is best to leave it in place for all kinds of diagnostic work, whether by film or by screen.

The writer has been surprised at the apparent carelessness with which he has seen the occasional radiologist subject patients to prolonged fluoroscopic observation in connection with fracture or foreign body surgery. Many medico-legal cases involving the use of the *x*-rays have resulted from such unwise prolongation of the screen control. This word of warning should not be amiss.

James T. Case.

FOR POSTERITY

THE portraits of men of achievement in any of the high professions should be painted by the worthy portrait painters of their country during their lifetime. While record of the actual work of our great men is carefully made and preserved the equally important contribution to history and posterity in the shape of a good portrait is more than often omitted. It is only when the famous doctor or engineer or philosopher or artist has passed on, that a regretful family seeks out a painter, presents him in most instances with a poor retouched photograph and begs for a lasting memorial of the departed husband, son or father who has labored faithfully for the advancement of his craft and the benefit of his community.

That photography has made tremendous strides in its artistic evolution no one will deny. Lighting, composition, fine printing are in many instances amazingly excellent, yet it is well to remember that after these attributes of a good photograph have been duly considered and accomplished, the mechanical click of the instantaneous shutter tells the final story.

The genius, the selective and observing brains of the well-equipped portrait painter are action of another and far more valuable caliber in setting down the essence, in the subtle personality, the speaking quality, to use a trite but none the less expressive term, of a man.

How valuable too, to the institutions where these men of achievement have worked, is a collection of their portraits; how much better than all the biographies does a good painting portray them and help not only as fitting adornment for the walls of their workshops, but as an inspiration to their younger successors in carrying bravely on!

The two group portraits of Dr. Gross and Dr. Agnew in their respective clinics painted by the great Thomas Eakins of Philadelphia are interesting. Sargent's splendid group of Drs. Osler, Welch and Kelly of Johns Hopkins also comes to mind as does the recently painted group of doctors, ex-internes of the Lankenau Hospital, Philadelphia, surrounding Dr. John B. Deaver in his operating room, painted by Albert Sterner.

What finer gesture can be made by a college, medical society, hospital group or group of public spirited citizens than to have done, in oils by worthy painters, portraits of their physicians who have achieved unusual distinction! T. S. W.

ABORTION AND CONTRACEPTION

To the Editor:

In response to your invitation to comment on the editorial by Dr. Gordon in your March issue, in which the doctor unhesitatingly condemns abortion and contraception, I wish to take issue with his stand. In Ohio we have a statute in accordance with which if three physicians concur in a decision that abortion is necessary to save the life of the mother, the production of such an abortion is legal. Many abortions have been induced in this state in accordance with that statute, and I think the statute has the approval of practically all our physicians.

When Dr. J. Whitridge Williams published the paper to which Dr. Gordon refers, I heartily approved of his attitude and promptly wrote him a note of congratulation over his frank and sensible attitude, and I have seen no adverse comment except this editorial by Dr. Gordon.

On general principles, to which, however, there may well be exceptions, as in some of the cases reported by Dr. Williams, abortions should be done only to save the life of the mother, or at least to save that life from extreme jeopardy. When it comes to contraception, however, conditions are different, and the judgment of the physician has a much wider range. I firmly believe in contraception in cases of mental and physical diseases which would probably be transmitted to the offspring, or in which pregnancy would be apt to affect deleteriously the life or health of the mother. There are many other conditions which demand humane and intelligent consideration, and the decision as to advising contraception can be reached only after the exercise of well-balanced judgment.

I feel that every woman has certain personal rights in the matter of maternity, and I have never had any sympathy with surgeons who refuse to side-step the tubes to prevent future pregnancies in cases of cesarean section. I have made many such sections, but invariably I ask the woman and her husband as to their desires for more children by the same method, and if they express a wish that there shall be no more pregnancies I always side-step the tubes. In only two instances has the mother expressed herself against it: one of them had the tubes side-stepped after the second cesarean section, but the other still persisted in their preservation at the second section, but years have gone by and she has had no further pregnancies and is now well past that possibility.

There is one church which we are told absolutely forbids any interference with the life of the fetus, or in any procedure that shall prevent pregnancy; and I can easily understand how a physician brought up in the tenets of that church might oppose both procedures, but aside from that I see no reason why the wishes of the husband and wife should not be paramount. If any of my patients in whom I have thus side-stepped the tubes have regretted it, I have not heard of it.

J. F. Baldwin, M.D.
Columbus, Ohio.

FOUR INDEX NUMBERS A YEAR

This number marks the completion of the first volume of The American Journal of Surgery since the increase in size in January, 1930. It comes to 1370 pages. As this makes a volume of unwieldy proportions, we will, beginning with July, 1930 (Volume IX), publish four volumes a year, instead of two, issuing indices with the September, December, March and June numbers. Consultation with librarians and subscribers has shown that this is a more desirable and practical method than to

attempt to continue only two volumes a year. It is found that an increasing number of subscribers are keeping bound files of The American Journal of Surgery. These present a record of the surgical progress of the day, and the extra indices are published in the interest of our subscribers.

THE AMERICAN ASSOCIATION FOR THE STUDY OF GOITER

ANNUAL MEETING 1930

JULY 10 AND 11, SEATTLE, WASH.

JULY 12, TACOMA, WASH., AND MOUNT RAINIER

Thursday, July 10

8:00—Registration, etc., at the Olympic Hotel

9:30—Address of Welcome. Hon Frank E. Edwards, *Mayor of Seattle*

10:00—President's Address. Dr. E. R. Arn, Dayton, Ohio: Auricular Fibrillation Associated with Hyperthyroidism

10:30—John Martin Askey, Los Angeles: Blood Pressure and Goiter, a Preoperative and Postoperative Study

10:45—E. P. Sloan, Bloomington, Illinois: Anatomy of the Thyroid Region

11:00—Wm. Engelbach, Santa Barbara, Calif.: Simpler Diagnostic Methods of Endocrine Disorders

11:15—W. O. Thompson, Chicago: Course of Exophthalmic Goiter Following Subtotal Thyroidectomy

11:30—LeRoy Long, Oklahoma City: Some Remarks on the Management of Hyperthyroidism

11:45—Raymond J. Millzner, Oakland, Calif.: Parathyroid Studies

1:00—Leo P. Bell, Woodland, Calif.: Tumors of the Neck Simulating Goiter

1:15—C. A. Roeder, Omaha, Neb.: The Third Type of Toxic Thyroidism

1:30—Martin B. Tinker, Ithaca, N. Y.: Thyroiditis

1:45—J. Rudolph E. Sievers, Butte, Mont.: Influences Bearing Upon End Results in Goiter Treatment

2:00—E. P. McCullagh, Cleveland: Relationship of Carbohydrates to Calcium and Phosphorus Metabolism in Tetany

2:15—Gordon S. Fahrni, Winnipeg, Canada: Effects of Thyroidectomy Upon Pregnancy

2:30—William J. Kerr, San Francisco: Necrosis of the Heart and Liver in Thyrotoxicosis

2:45—General Discussion

4:00—Ralph R. Wilson, Kansas City, Mo.: Effects of Thyroid Activity Upon the Female Organism

4:15—John S. Helms, Tampa, Fla.: The Incidence of Goiter in the Southeastern States and Its Possible Significance

4:30—H. W. Riggs, Vancouver, B. C.: Adenomatous Goiter

4:45—General Discussion

7:00—Boat Ride

Friday, July 11

8:00 to 10:00—Surgical Clinics. Doctors Brien T. King, A. J. Bowles, C. C. Tiffin, J. Tate Mason, and J. C. Moore.

11:00 to 12:00—Henry S. Plummer, Rochester, Minn.: Thyroid Therapy

1:00—S. D. Van Meter, Denver: Classification of Goiter

1:15—Thomas M. Joyce, Portland, Ore.: Preparation of Thyroid Patients for Surgery

1:30—Lewis M. Hurxthal, Boston: Cardiovascular System in Goiter

1:45—Norris W. Gillette, Toledo, Ohio: Complications Following Thyroidectomy

2:00—Thos. O. Burger, San Diego, Calif.: Local Anesthesia with Adjuncts for Thyroidectomy

2:15—Allen Graham, Cleveland: Malignant Tumors of the Thyroid, a Study of End Results

2:30—Willard Bartlett, Jr., Saint Louis: Additional Method of Determining the Operative Risk in Goiter

2:45—J. Earle Else, Portland, Ore.: Prevention of Postoperative Recurrences in Thyroidectomy

3:00—Joseph L. DeCourcy, Cincinnati: Rare Tumors of the Thyroid Gland

3:15—General Discussion, Opened by Wm. O. Sweek, Phoenix, Ariz.

3:45—Robertson Ward, Jessie L. Carr, San Francisco: Malignant Goiter

4:00—Addison G. Brenizer, Charlotte, N. C.: Hyperthyroidism and Hypothyroidism Following 2500 Thyroidectomies

4:15—Lindon Seed, Chicago: Differential Diagnosis of Thyrotoxicosis and Neurosis

4:30—General Discussion

7:30—Banquet. Address by Karl F. Meyer, San Francisco.

Saturday, July 12

8:00 to 9:00—Diagnostic Clinic: Arnold S. Jackson, Madison, Wis.

9:00—Earle Drennen, Birmingham, Ala.: Analysis of 125 Operations on the Thyroid Gland

9:15—O. P. Kimball, Cleveland: Relation of Endemic Goiter to Mental Deficiency

9:30—Warren H. Cole, Saint Louis: Experimental Production of Pathological Changes in the Thyroid by Infective and Chemical Means

9:45—Henry J. Vandenberg, Grand Rapids, Mich.: How Long Is a Toxic Goiter Toxic?

1:00—Luncheon at Tacoma, with Pierce County Medical Society. E. Starr Judd, Rochester, Minn.: Treatment of Peptic Ulcer and Gallbladder Disease Complicated by Hyperthyroidism T. Addis, San Francisco: Do Cases of Bright's Disease Have Disturbances of the Thyroid Gland? Dinner at Paradise Inn, Mount Rainier.

"ADDISON'S DISEASE"

A COLLEAGUE of Richard Bright at Guy's, known as an inspired pathologic lecturer and diagnostician rather than as a successful practitioner, Thomas Addison was born near Newcastle in 1793. He graduated in medicine at Edinburgh in 1815, after which he settled in London.

We are told his manner was repellent and haughty. However, this aloofness was a defensive mechanism to hide a shy and excessively sensitive inner-man. Because of these characteristics he never had a large practice. Perhaps it is well that this was the case. To his students and his hospital he gave unstintingly of the best that was in him.

He was decades ahead of his time in that he had little use for the polypharmacy of those days. It is said that he often left his patients forgetting to prescribe for them. However, "Addison's pill," composed of calomel, digitalis and squills, in certain syphilitic conditions is still prescribed.

Addison was the first to prescribe static electricity in the treatment of spasmodic and convulsive diseases (1837), and with John Morgan wrote the first book in English on the action of poisons on the living body (1829). He published an excellent account of appendicitis in 1839.

In 1837 he became physician to Guy's Hospital. His chief researches were on pneumonia, phthisis and especially on the suprarenal capsules. He wrote on disorders of the female, and, with Dr. Bright, "Elements of the Practice of Medicine."[1]

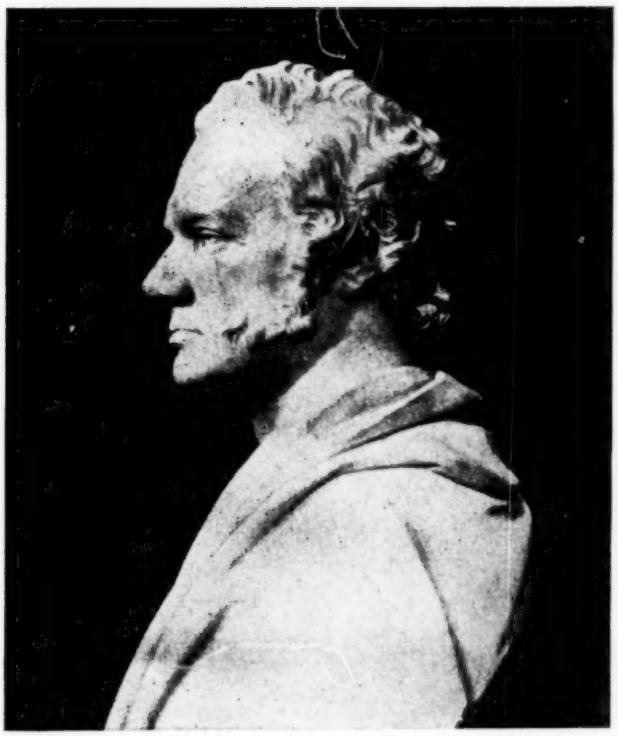
In 1849 Addison read a paper[2] describing pernicious anemia and diseases of the suprarenal capsules. These clinical articles were at a later date expanded in his great monograph, "On the Constitutional and Local Effects of Diseases of the Suprarenal Capsules."[3] At the time this work was merely a scientific curiosity. It is now looked upon as epoch making.

Addison described a constitutional affection manifesting itself in an exaggeration of the normal pigment of the skin, asthenia, irritability of the gastrointestinal tract, and weakness and irregularity of the heart's action; these symptoms being due to loss of function of the suprarenal glands. It was Trousseau who first proposed to call this suprarenal syndrome "Addison's Disease," by which it has been known to date.

Addison died at the age of sixty-seven years on June 29, 1860.

T. S. W.

[1] Vol. 1, 1839.
[2] *Lond. Med. Gaz.*, 32: 517, 1849.
[3] Lond., 1855.



THOMAS ADDISON

[1793-1860]

The American Journal of Surgery
N. S. Vol. VIII, June, 1930

[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

STUDIES IN PALEOPATHOLOGY XXV

HYPERTROPHY IN THE SACRUM OF THE SABRE TOOTH, PLEISTOCENE OF SOUTHERN CALIFORNIA

ROY L. MOODIE, PH.D.

SANTA MONICA, CALIF.

OSSEOUS hypertrophy, as seen in the skeletons of fossil vertebrates, may indicate a variety of conditions. Pachyostosis is one of these and represents a non-pathological heaviness developed in the skeleton of vertebrates in response to a number of causes. This phase of paleontology is well known and has been so recently discussed that nothing more need be said about it here. Hypertrophy of bone accompanying a wide range of pathological conditions is also well known so it only remains for us to locate the present example of osseous hypertrophy in its proper place.

Examination of the figures will show that there is a necrosed area on the dorsal surface of the sacrum, to the right of Figure 1. I am going to state that, probably, the tiger to whom this pelvis belonged received a stab wound from the sabres of another tiger which having become heavily infected, resulted in the hypertrophic changes which are so evident in the specimen. The condition seen is thus interpreted as an example of osseous hypertrophy following a violent trauma, subsequently infected. The example is quite unique and forms a striking object in the series of pathological pelves of the *Smilodon*. The last two lumbar vertebrae, firmly ankylosed to the sacrum, indicate that a slight scoliosis had developed as one of the changes due to the injury. The most striking change is the ossification of all the sacral ligaments, dorsal and ventral, accompanied by hypertrophic changes in the sacral and lumbar vertebrae themselves. I have sought eagerly for hypertrophic changes due to endocrine disturbances, but so far without success. A gigantic skull of a lion, one-third larger than any other lion skull in the collection, suggests pituitary changes; but a careful examination of the endocranial cavity is yet to be made.

The normal sacrum of the sabre-tooth has three vertebral segments, measuring in three specimens from 110 to 120 mm. in length. There are 1086 sacra of young and adult sabre-tooth tigers in the collections of the Los Angeles Museum, all of which I have examined. Only fourteen of them are pathological, and the present specimen is the most extensively changed of any of them. Even in old individuals

the sacrum is not preserved with the pelvic rami. Only three specimens retain this association and all of them are pathological. While there is some range in the length and width of the sacra none save the one here described exhibit hypertrophy. Five of them exhibit the phenomenon of sacralization which will be described in another paper.

Except for the slight pathological changes in the crest of the right ramus and its firm coossification with the sacrum and last lumbar, the pelvis is unchanged. A comparison with several pelves shows a considerable variation in size. The acetabular cavities are unaffected. Pathological conditions in the pelvis of the sabre-tooth will be described elsewhere. Changes due to disease are quite extensive in one specimen.

The most striking feature of the dorsal surface of the hypertrophied sacrum (Fig. 1) is the ossification of the vertebral ligaments, especially the Ligamenta interspinale, supraspinale, intertransversarum.[1] The differences between the right and left sides in the ossification of the ligaments may be explained by the presence of the abscess represented by the necrosed area on the right. There are a few blunt exostoses accompanying the ossification of the ligaments. The coossification between the pelvis and the sacrum is very intense.

Fig. 1. Dorsal aspect of hypertrophied sacrum and pelvis of young, though mature, sabre-tooth (*Smilodon californicus*), showing intense ossification of ligaments, following injury, effects of stab wound received in right pelvis. Two lumbar vertebrae are firmly united to sacrum by ossified ligaments. Length of right pelvic ramus 320 mm. Left ramus was broken and part of it is lost. Length of vertebral mass, as preserved 160 mm. Sacrum proper is broken through middle of second segment. There is such firm coossification between sacrum and pelvis that a post-fossilization break took away part of each. Pelvis is otherwise unaffected. (Specimen in the Los Angeles Museum.)

[1] See Studies in Paleopathology. *Ann. Med. Hist.*, 8: 78; 288, 1926, for explanatory diagram.

Ossification of the ligaments of the lumbar vertebrae is not intense. The slight scoliosis is to be explained by the ossification of the ligaments on the left.

The sacrum had been broken, possibly during excavation, through the middle of the second sacral segment. The broken surface has a transverse diameter of 60 mm., and it is hypertrophied to more than one-third larger than normal. The neural canal is obliterated. This was not serious since, like the modern bob-cat, the sabre-tooth had a very short tail, and the last part of the sacrum contained a sort of cauda equina. The hypertrophy is ascribed to changes following upon a severe injury, subsequently heavily infected.

The ventral surface of the specimen (Fig. 2.) shows intense ossification of the ligaments, largely of the left side. The left transverse process of the last lumbar had been broken during life, and healed.

I think the appearances of the lesions indicate that suppuration had ceased before death. Except for a period of intense pain, following the infection, and some stiffness in the pelvic muscles the activities of the animal would not have been interfered with by the results of the injury.

Fig. 2. Ventral aspect of same pelvis showing the ossification of ligamentum longitudinale. Anterior surface of lumbar vertebra is unaffected, so that pathological condition may be spoken of as spondylitis deformans.

BOOK REVIEWS

The Dramatic in Surgery. By Gordon Gordon-Taylor, O.B.E., M.A., F.R.C.S. 88 pp. 40 illus. N. Y., William Wood & Co. 1930.

The book with this most attractive title is based on an address delivered in Manchester, February, 1928. The contents, like the title, are delightful. There are forty splendid illustrations. As the author well points out "it is often for those morbid conditions which are *infinitely little* rather than for those which are gigantic or colossal . . . that the *most expert*, . . . but perchance the *least dramatic* surgery is really required," and further that: "perhaps Caesarean section proves the greatest source of attraction . . . though, of course not nearly requiring as much expertness as many an operation that would attract little attention on the part of the uninitiated." The author's own dramatic instinct is indicated by his calling the foreign bodies, which are removed from the *corpus humani*, "trophies," and his reference to "surgical megalomania."

That "the dramatic in surgery may be attained alike by the *elaboration* or by the *simplicity* of the requirements which are regarded as essential to the art," should be impressed upon all students. Simplicity in surgery, as well as in all other arts, is undoubtedly the greatest and most difficult of achievements.

As the most modern touch, he mentions that "as a means of rendering 'first aid' the aeroplane may be valuable." Considering the arrival of the surgeon in an aeroplane as part of "the dramatic in surgery" is in itself rather good theatre.

Humorous instances are not overlooked by

our author and here, as elsewhere, the ubiquitous Scotchman is quoted: "A Scotchman walking down Charing Cross Road on the way to Charing Cross Hospital to have the hydrocele tapped, saw a sixpence on the pavement and stooped to pick it up; he ruptured the hydrocele and was brought to Middlesex Hospital instead."

It must not be thought for a moment that this book is only for amusement or pastime. It contains many an axiom that may well be taken to heart by even the most experienced surgeon, such as "This at least is certain: the most thorough co-operation of surgeon, physician, radiologist, and laboratory worker is necessary to decide the need of surgery and to determine the appropriate character of the operation to be performed." A small book, but well worth while.

THE ACTION OF MUSCLE. By Sir Colin Mackenzie, M.D., F.R.C.S., F.R.S., (Edin.), Ed. 2, 288 pp., 100 illus., N. Y., Paul B. Hoeber, Inc., 1930.

This is the second edition of this widely read work.

Since the Great War greater attention has been paid to the importance of muscle function, whether in the consideration of health or disease, than formerly. Physiotherapy today plays an important part in every modern hospital. In this new edition a special section has been added dealing with the erect posture. The author dealt with these principles in his presidential address before the Section of Zoology at the Australasian Science Congress in 1928.

After the introduction the book considers Principles, The Shoulder Region, The Biceps Brachii and Brachialis; Musculocutaneous Paralysis, Median Nerve Paralysis, Ulnar Paralysis, Musulo-Spiral Paralysis, The Muscles of the Thigh, Muscles Acting on the Leg, Muscles Acting on the Foot, The Muscles of the Toes, Muscles of the Spine, Muscles Which Move the Neck, Muscles Which Move the Back and Loins, Anatomical Considerations in Joint Fixation, The Muscles of Respiration. The book is well indexed and contains 100 illustrations.

Any doctor will profit by reading this important work but we specially recommend it to surgeons and neurologists.

NEUE DEUTSCHE CHIRURGIE. Begrundet von P. von Bruns. Herausgegeben von H. Küttner in Breslau. 44 Band. DIE CHIRURGIE DER BASEDOWSCHEN KRANKHEIT. Von Dr. Heinrich Klose, Prof. der Chirurgie an der Universität Frankfurt Am Main; Direktor der Chirurgischen Abteilung des Stadtischen Krankenhauses der Freien Stadt Danzig. 675 pp. mit 273 Teils Farbigen Abbildungen. Stuttgart, Ferdinand Enke, 1929.

45 Band. DER WUNDSTARRKRAMPF BEIM MENSCHEN. Von Dr. Arthur Buzello, Prof. an der Universität Greifswald. 276 pp. mit 51 Textabbildungen. Stuttgart, Ferdinand Enke, 1929.

46 Band. CHIRURGIE DER MILZ. Von Prof. Dr. H. Hirschfeld, Berlin und Prof. Dr. R. Mühsam, Berlin. 286 Seite mit 32 Teils Farbigen Textabbildungen. Stuttgart, Ferdinand Enke, 1930.

47 Band. DIE HÄMOPHILIE. Von Prof. Dr. H. Schloessman, Direktor der Chirurgischen Abteilung der Augusta-Krankenanstalt Bochum. 321 pp. mit 60 Textabbildungen. Stuttgart, Ferdinand Enke, 1930.

Four new volumes of the famous "Neue Deutsche Chirurgie" are before us and as usual each volume is a complete and up to date monograph on its particular subject.

Klose's book on "Basedow's Disease" is the largest of the four and especially well illustrated. With a controversial subject in hand, the author had a difficult time of it. In his Preface he explains that the book does not suit him completely, but that he has acceded to the pressure put upon him by both the editor and the publisher to produce the volume at once. The reviewer feels that the author is entirely too modest and that the book may well be accepted as a comprehensive and thoroughly scientific review of the present status of the etiology, pathology and treatment of this disease. His plea that the surgeon should always work in cooperation with the internist in the treatment of this condition is one that every American surgeon may well take to heart.

The volume by Buzello on Traumatic Tetanus covers, as the author states, one of the most important and terrible wound infections with which we have to deal. Tetanus is considered as a bacteriological infection and also as a nervous disease, so that the book will also interest the clinician. The author feels that as tetanus usually follows wound infection, its treatment will continue to be in the hands of the surgeon, especially as such an infection involves important and serious surgical operations. Here again, as in the other volumes of the series, the necessity for cooperation between the physician and surgeon is particularly stressed, even though the ultimate treatment is felt to be entirely surgical.

Hirschfeld and Mühsam take up the present status of the surgery of the Spleen. It is interesting to note that whereas this volume on

the Spleen has 286 pages, the volume on this subject in the "Deutsche Chirurgie" published in 1890 contained only 57 pages. In the last forty years progress has indeed been made in the surgery of the spleen, and its removal is today advised for many other conditions beside tumors of the spleen. Here again, as in the volumes mentioned here, the authors make a plea for the utmost cooperation between the surgeon and the internist. Up to the minute, thorough and profusely illustrated, this volume will be invaluable for reference to German reading surgeons.

Hemophilia is discussed in 306 pages by Prof. Schloessmann, who has had unusual opportunities for research in this condition during many years at Württemberg. This work contains an unusual amount of personal research and conclusions drawn therefrom, combined however with a complete study of the international literature on the subject. Like our American writers, the author feels that most progress in the prevention of this disease will come from a more thorough study and knowledge of eugenics. The book is fully up to the standards of the other volumes of this series.

All the volumes of the "Neue Deutsche Chirurgie" contain splendid bibliographies. It is to be regretted, however, that each volume has not a detailed index. They all have comprehensive table of contents, but a complete index would facilitate ready reference.

Modern Otology. By Joseph Clarence Keeler, M.D., F.A.C.S. 878 pp., 90 original illus. and 15 colored plates. Phila., F. A. Davis Co., 1930.

There is nothing so difficult to review fairly as a textbook. It should bear the stamp of the author's individuality; at the same time it should not feature any of his hobbies too strongly. Like other methods of teaching, it is not a question of whether one agrees with the particular method used, but of the success attained in getting the subject across to students. Dr. Keeler recognizes this difficulty when he states that "During many years of otologic practice and preaching . . . I have always felt that the existing textbooks were unsatisfactory. Though their good points outnumbered their faults . . . " And he adds "When, occasionally, I voiced this dissatisfaction . . . the advice I usually received was: 'Write a book yourself.'" Now, of course, his own book must submit to the same attitude on the part of others. The subject is completely covered, the illustrations are well selected and well reproduced; in short we have before us another good, conventional, textbook. That it will appeal to many teachers and students is certain; that it will not suit everybody is equally certain. Were there not differences of opinion there would be occasion for only one textbook on each subject, a condition that is often prayed for by teachers, students and publishers. On the other hand, the very variety of books, forcing critical selection, undoubtedly makes for better teaching, better textbooks, and better practice. Every otologist will want this book if only for comparison with others. The man having only one book on otology will not go far wrong if he selects Keeler's "Modern Otology," and the same could be said of several other books on the subject.

A Shorter Surgery. A Practical Manual for Senior Students. By R. J. McNeill Love, M.B., M.S. (Lond.), F.R.C.S. (Eng.) Ed. 2, 389 pp., 74 illus., including 31 plates (one colored). N. Y., William Wood & Co., 1930.

This book lives up to its title: "A Shorter Surgery." For a quick freshening up on the general subject for examination or otherwise this work should be valuable. The author states that it "is an attempt to condense and crystallise the more important principles of surgery for the benefit chiefly of the student." Whether it is too short for the student is a problem that the individual instructor will have to decide for himself. It appeals to the reviewer more as a quick reference for the experienced surgeon. In any circumstances it can be considered only a supplement to larger textbooks. The book is well illustrated.

Therapie und Therapeutik: Ein Mahnruf an Ärzte, Kliniker und Pharmakologen. By Prof. Dr. I. Boas. vi + 79 pp. Verlag von S. Karger, Berlin, 1930.

It is encouraging to note that the great German precisionistic movement in science which swept through medicine during the last fifty years has at last embraced therapeutics. Dr. Boas makes an impassioned plea for scientific therapeutics studied under controlled conditions as immediately necessary if medicine is ever to get over the barren wastes of a traditional therapy. The plea is timely for Germany, where commercial exploitation is making a mockery of the treatment of disease. Thanks to the "therapeutic nihilism" of Osler and the weighty influence of the Council on Pharmacy and Chemistry of the American Medical Asso-

ciation, American medicine has long been developing a healthy interest in real scientific therapeutics. Dr. Boas might have used our experience to advantage in his thesis. At any rate, we may watch the effect of his "warning" with sympathetic interest.

STRAHLENBEHANDLUNG DER WEIBLICHEN GENITALCARCINOME, METHODEN UND ERGEBNISSE (Radiation Therapy of Female Genital Carcinoma, Methods and Results). By Prof. Dr. Friedrich Voltz, Dir. Radiotherapy Section, Gynecol. Clin. Univ. of Münich, with foreword by Geheimen Rat Prof. Dr. Albert Doderlein, Dir. Gynecol. Clin. Univ. of Munich. 57 colored illus., 8 colored charts and 40 tables. Berlin, Urban & Schwarzenberg, 1930.

This 206 page work represents a real contribution to our present knowledge of methods and results of radiotherapy in the treatment of genital carcinoma of women. The work done in this field by the Doderlein Clinic of Munich is well known to every gynecologist and radiologist doing gynecological radiotherapy. Professor Doderlein made his first very important report on this subject from his clinic in 1913 before the International Congress of Medicine in London.

There is a brief chapter on the development of radiation therapy of female genital carcinoma. The second chapter deals with the effort being made by the Hygienic Section of the League of Nations to forward the progress of the campaign against carcinoma. In fact this book really represents a report by Professor Voltz, as a member of the sub-committee of the League of Nations, Hygienic Department, appointed to investigate genital carcinoma in women.

The author gives a classification of carcinoma of the female genital tract, with many beautiful colored illustrations of the histological appearances, followed by a description of the roentgen apparatus for deep roentgen therapy, the apparatus for measuring dosage, and a description of the necessary supply of radium and radium applicators for gynecological radiotherapy.

The general method of treatment which is pursued at present in the University of Munich Gynecological Clinic is as follows:

First day: Examination of the patient, taking of the history, and study of the various clinical findings, and marking out of the various parts of entry ready for the treatment.

Second day: Radiation over the hypophysis. This was first suggested by Henry Hirsch and since the year 1923 has been part of the routine treatment of all carcinoma patients in the Munich Clinic. The idea is to sensitize the carcinoma. The author has seen no disadvantage from this treatment; on the contrary he is quite convinced that the general condition of the patient has been very much improved during the radiotherapy. Two lateral fields are employed, 6×8 cm. The focal skin distance is 30 cm. and the treatment amounts to 30 per cent of the erythema dose at 200 kv., with 2.5 ma. and 1 mm. of copper filter. This treatment is given only once at the time of the first series of treatment.

Third day: The patient is given an injection of "Dextrocid," following which the physician goes ahead with the general radiotherapy to the pelvis. This "Dextrocid" injection is an intravenous administration of glucose, which has been done routinely in all their cancer cases since 1927. It also was introduced by Henry Hirsch in Hamburg. The author claims to have seen beneficial results following this injection.

Fourth day: The fourth day is rest day. Sometimes more than one day is necessary.

Fifth day: On the fifth day is done any diagnostic work necessary, such as the excision of specimens, or of extrafoliating tissue, or the cauterization or cataphoretic treatment as may be necessary.

Sixth day: The sixth day is rest period which may be extended if necessary.

Seventh day: Glucose injection and treatment with radioactive substances.

Eighth day: The eighth day is rest day.

Ninth day: Discharge of the patient.

The second series of treatment occur after an interval of eight weeks from the day of the discharge of the patient. The third series may sometimes be given after another interval of eight weeks.

The result can hardly be reported in a review. The authors not only give their own results but they summarize the results published from numerous sources both in Europe and in America. They conclude by expressing the hope that this work, with its published results, will further emphasize the declaration of Professor Doderlein that the radical operation for uterine carcinoma is no longer justifiable.

Concluding chapters are translations of reports from the Radium Hemet of Stockholm by Heyman and from the Radium Institute in Paris by Lacassagne and by Regaud.

Traité de Radiodiagnostic, Squellette en General (Tete-Rachis-Bassin-Membres). By G. Chaumet, Ass. Prof., Val-de-Grace, Paris. 202 figs. Paris, Vigot Frères, 1930.

This 350 page treatise on the *x*-ray diagnosis of bony lesions is the result of the author's belief that it is well to bring together periodically the well established facts and from them to take stock of our resources. To rejuvenate old ideas, to eliminate certain lines of effort which seem useless, to moderate enthusiasm for methods or theories which appear to be too popular, to offer a critical analysis of results in the light of experience, and to make a slight personal contribution, these are the reasons given by the author for his book.

All progress is achieved at a price. The perception of bones through soft tissues carries with it a certain number of illusions: the deformities, enlargements, superposition of shadows due to the conical projection of the *x*-rays, all the optical phenomena which lead to error. It is the duty of the radiologist to profit by investigation in new improvements of technic and by the employment of new positions and angles of incidence.

The patient presents numerous individual problems. The reading of the radiographic films cannot be correctly done without a certain familiarity with pathology. But even the best possible film cannot be judiciously interpreted by the most distinguished physician in the world if he lacks the necessary training.

The task of the medical radiologist rests upon a triple foundation: (1) Adequate radiographic technic; (2) abundance of experience in interpretation; (3) a broad medical training.

After a brief consideration of general principles and a chapter on the radiographic appearances produced by various morbid processes in bone, the author proceeds to a regional discussion of bony lesions as revealed in the radiographic films. There is an excellent outline for the study of bones. Numerous illustrations add value to the suggestions as to improvements of technic. Various positions are advised which, though not in common use, merit careful consideration and more frequent employment.

Gallbladder Disease, Roentgen Interpretation and Diagnosis. By David S. Beilen, B.S., M.D., Roentgenologist, Augustana Hospital, Chicago. St. Paul, Bruce Pub. Co., 1929.

There is much about this work to commend it. There are large, well reproduced illustrations, rich in radiographic detail. The text, though meager, presents the clinical and pathological facts in order, with the *x*-ray evidence as a series of films for each case. The work should prove helpful in the interpretation of oral cholecystography. The author dismisses with a few words the intravenous method as of little importance, although Graham and his associates, after many thousands of trials of both oral and intravenous administration of the tetraiodophenolphthalein, still emphatically advise the intravenous route as more reliable. As an exposition of the views of one who is entirely satisfied with the oral method and claims an unusually high percentage of operatively proved accuracy, this work is an admirable product.

Taschenbuch der Medizinischen Rontgen-und Radium Technik. By Dr. Phil. Gottfried Spiegler and Dr. Phil. Albert Fernau, Leiter der Rontgentechnischen Versuchsantalt am Zentralrontgeninstitut des Allgemeinen Krankenhauses in Wien and Privatdozent fur medizinische Physik und Chemie des Radiums an der Universität Wien, respectively. 330 pp. and 63 illus. Wien, Julius Springer, 1930.

An excellent brief introduction to the practice of radiography and radium therapy. The book abounds in tables, formulae and rules for protection. There is much detailed information regarding minor points of technic included in this systematic consideration of the physical and practical bases of radiologic diagnosis and therapy.

X-ray Technology, The Production, Measurements and Applications of X-Rays. By H. M. Terrill, Ph.D., Assoc. in Physics, Inst. Cancer Research and C. T. Ulrey, Ph.D., Research Physicist, Westinghouse Lamp Co. N. Y., D. Van Nostrand Co., Inc., 1930.

The authors state that in preparing this book it has been their purpose to present the practical rather than the theoretical aspects of *x*-ray measurements as they are inevitably encountered by all who choose to work in the *x*-ray laboratory. Much of the contents is devoted to problems common to all *x*-ray laboratories but particular attention has been given to the quantitative measurements involved in roentgen therapy and in the application of the *x*-ray to industrial problems. A considerable portion of the contents may be properly classified as *x*-ray engineering.

Properties of moving electrons are discussed in 25 pages, *x*-ray tubes and their operation in 50 pages, generators of high voltage in 20 pages. Pages 99 to 211 discuss measurements of voltage and current, ionization, total energy,

wave-length and biological problems. The remaining 40 pages are devoted to *x*-ray analysis and radiography, especially in its industrial applications.

This is a very readable, interesting book and well worth acquisition by all interested in radiology.

Yourself, Inc., The Story of the Human Body. By Adolph Elwyn 332 pp., 71 illus., N. Y., Brentano's, 1930.

The title of this book will undoubtedly appeal to those whom it is intended to attract. To the reviewer the sub-title "The story of the Human Body" is much more attractive but "wise-crack" titles seem to be the order of the day and if "Yourself, Inc." will draw additional attention to the story of the human body so much the better for the readers, for the author, the publishers and for the medical profession. The story is well told, accurate and definite, and may be recommended without compunction to every intelligent layman. Scientific facts are put before him in language that he will readily understand but which, at the same time, assume more common sense on the part of the reader than many authors of so-called "popular medical books" are in the habit of accrediting to him. The first chapter, regarding the title of which there seems to a difference of opinion between the caption writer and the index maker (for it is indexed as Looking Backward and the running head reads Looking Back), is a delightful fifteen-page abstract of the History of Medicine to the time of Vesalius and Harvey. The other chapters in turn are headed Introducing the Body, Concerning the Cell, The Living Membranes, The Building Trades and Their Strange Associates, The Transport Workers, The Pump and Engine Workers, The Engines of the Human Body, The Transportation System, The Food and Fuel Industries, The Utilization of Air, The Disposal of Garbage, The Central Government, The Central Government (continued), The Regulation of Home Activities, Concerning Reproduction, Looking Forward. The author is very apt in his ability to draw an analogy between the workings of the human body and those of machinery.

Chirurgie des Kindesalters. By Prof. Dr. R. Drachter and Dr. J. R. Gossmann. 1047 pp., 714 illus., Leipzig, F. C. W. Vogel, 1930.

This new edition has been brought entirely up to date as is evidenced by the mention of Intravenous Pyelography. Incidentally, attention is called in the Preface to the fact that the portion on the urinary tract has been completely revised. The illustrations, both colored and black and white, leave nothing to be desired, and the text of over 1000 pages covers the subject thoroughly and without padding. It is an invaluable book on Surgery in Children that is indispensable to every German reading surgeon.

The International Medical Annual: A Year Book of Treatment and Practitioner's Index. Editors: Carey F. Coombs, M.D., F.R.C.P. and A. Rendle Short, M.D., B.S., B.SC., F.R.C.S. 609 pp., 61 pl., 54 illus., N. Y., William Wood & Co., 1930.

The annual return of the International Medical Annual is as welcome as are the flowers that bloom in the spring. Particularly for the man who "has not the time" to keep up with the voluminous literature appearing each month, this annual resume of the advances in medicine is a boon. With the death in April of last year of Dr. Wilde, its founder and editor, the new volume has been taken up by Drs. Coombs and Short, as editors, and the old traditions have been well preserved. Unusually well illustrated, this volume forty-eight presents, as do the previous ones, a compact and reliable review of the medical and surgical advances of the year.

Das fur und Wider der Chirurgischen Behandlung des Gallenleidens auf Grund der Erfahrungen an 800 Gallenoperationen. By Prof. Dr. Paul Zander, Chirurg des Elisabethenstifts in Darmstadt. 95 pp., 8 illus., Leipzig, Georg Thieme, 1930.

This eighty-nine page consideration of the perennial discussion concerning the medical and surgical treatment of gall bladder diseases presents both sides tersely. It comes, as do all such discussions, to the inevitable conclusion that the ideal situation is honest cooperation between the internist and the surgeon. The debate of this subject will continue for a long time and it is interesting to have this resume which really presents the present status of the case.

Merck's Index: An Encyclopedia for the Chemist, Pharmacist and Physician, Giving the Names and Synonyms; source, origin, or mode of manufacture; chemical formulas and molecular weights; physical characteristics; melting and boiling points, solubilities; specific gravities; medicinal action; therapeutic uses; ordinary and maximum doses; incompatibilities; antidotes; special cautions; hints on keeping and handling, etc., of the Chemicals and Drugs Used in Chemistry, Medicine and the Arts together with an Appendix containing: reactions of the more important alkaloids

and glucosides; characteristic reactions of acids, bases, metals, and salts; table of atomic weights; thermometric equivalents; specific gravity tables; metric conversion tables; and abbreviations. Ed. 4, 593 pp., Rahway, N. J., Merck and Co., Inc., 1930.

The third edition of this work was published in 1907 and it is explained that the delay in the publication of the fourth edition "was caused largely by the complete interruption of work on the book in both laboratory and editorial rooms during the World War." That the book lives entirely up to its title (as given here) is perhaps the best description that can be made of it and it is a high compliment indeed. It is believed that the hope expressed in the Preface "that it will meet the needs of the student, the apprentice, the young man on the threshold of his professional career" will be entirely fulfilled.

Obstetrics for Nurses. By Joseph B. De Lee, M.D., Ed. 9, 645 pp., 269 illus., Phila., W. B. Saunders Co., 1930.

The new ninth edition of De Lee's Obstetrics for Nurses deserves its widespread popularity.

In this edition many new features are included. The book has been carefully revised, completely reset and contains hundreds of new illustrations.

One can understand why this is the standard textbook in the large majority of training-schools for nurses. We might add, the type of general practitioner who has little time for quiet study and reading, who is rusty in the ordinary fundamentals of obstetrics, can read this book with mental and practical profit.

Gynecology for Nurses. By George Gellhorn, M.D., 275 pp., 145 illus., 12 mo., Phila., W. B. Saunders Co., 1930.

This is a book that should receive a welcome in every training-school for nurses. It is a good book from every angle. Dr. Gellhorn is especially fitted to have done this work. He has taught nurses for over twenty years. He knows what a nurse needs in the performance of her duties. In his gynecology for nurses as held to the sane and practical. He has left out the pseudo-scientific hokum, so noticeable in many works for nurses that are made into big books by unnecessary padding.

This book covers the subject. It is easily read. Any nurse is sure to grasp the thoughts conveyed by the author. It has 275 pages and 145 illustrations. We do not only cheerfully recommend it but we urge that it be in the hands of every pupil nurse.

Diseases of the Nose, Throat and Ear, Medical and Surgical. By William Lincoln Ballenger, M.D., F.A.C.S., and Howard Charles Ballenger, M.D., F.A.C.S. Ed. 6, 1138 pp., 583 engravings, 29 plates, Phila., Lea & Febiger, 1930.

The sixth edition of this work, revised by Howard Charles Ballenger, has become a formidable treatise of over 1100 pages and 600 illustrations. Drs. Tucker and Jackson have written the part on Bronchoscopy, Esophagoscopy and Gastroscopy. The chapter on the Labyrinth has been rewritten by Dr. Alfred Lewy. Other changes and revisions throughout the book have brought it up to date and it should retain for many years its position as one of the standard works of reference in its field.

Ueber Röentgenschaeden und Schaeden Durch Radioaktive Substanzen. By Priv.-Doz. Dr. Wilhelm Flaskamp, Gynecological Clinic, Univ. of Erlangen. 365 pp., 94 illus., 30 half-tone plate illus. and 6 tables. Berlin, Urban und Schwarzenberg, 1930.

Just what we have been looking for! A collection of all the known facts concerning the possible damage resulting from the use of roentgen rays and other radioactive sources. This mine of information will undoubtedly find its way very soon into the library of every member of the legal or medical profession who has an active interest in *x*-ray damages and the legal action which so often attend them. From the scientific point of view the work is also a rich repository of facts gleaned from the literature of the world. The book is illustrated with numerous photographs, some of them series of pictures of the same case taken during the life of the patient and therefore constituting a document of unusual worth.

The first section deals with a brief historical statement, the various theories relating to the mechanism of damage to the skin from *x*-ray exposure, and a very detailed study of the biological and histological manifestations of roentgen erythema and dermatitis, treated from every possible point of view. Other high lights are sections on roentgen carcinoma, roentgen ulcer, telangiectasis, and the prophylaxis and treatment of roentgen dermatitis.

The second section concerns damage from *x*-rays to the deeper tissues and the internal organs, including in turn a consideration of the subcutaneous tissues, muscles, bones and joints, the teeth, serous membranes, the lungs, larynx, intestines, the urinary tract, brain and central nervous system, the eyes, the glandular organs (thyroid, thymus, hypophysis, pan-

creas, liver, etc.). Liberal space is given to the effects upon the generative organs of men and of women, and especially the influence of irradiation upon pregnancy. Final sections are devoted to the blood and the blood-building organs, and general roentgen reactions. Very full lists of references to the literature terminate this very important contribution to the literature of radiation therapy.

DIE RÖNTGEN-LITERATUR Autoren- und Sachregister Patentliteratur. Herausgegeben von Dr. Hermann Gocht. VIII Teil, 1927, 350 pp. IX Teil, 1928, 384 pp. Stuttgart, Ferdinand Enke, 1928, 1929.

The eighth and ninth volumes of Gocht's annual bibliography of radiological literature present a picture of the work done in radiology in 1927 and 1928. The literature of all languages seems to be thoroughly covered. Everyone interested in radiology owes a debt of gratitude to both Dr. Gocht and his publishers for making available annually in a handy volume a bibliography of this increasingly important subject.

A fairly careful check-up reveals few errors or omissions. The book arouses in the reviewer a wish that a similar bibliography could be published on every specialty of medicine. The finding and checking of references would indeed be simplified.

RÖNTGENUNTERSUCHUNGEN AM INNENRELIEF DES VERDAUUNGSKANALS von Dr. Hans Heinrich Berg. Ein Beitrag zur klinischen Roentgendiagnostik Insbesondere Von Entzundung, Geschwur Und Krebs. 193 illus., Leipzig, Georg Thieme, 1930. Price, paper 27 m., bound 29 m.

One of the evolutions which has been taking place in diagnostic radiology concerns the more accurate study of the lining of the alimentary tract. This was first accomplished, as is well known, by opaque materials introduced into the stomach, accomplished in 1897 by Cannon and Williams in Boston and then 1908 popularized by Rieder in Munich. This method undoubtedly opened up an entirely new field in diagnostic radiology and has resulted in an accuracy of diagnosis exceeding that of all the other laboratory methods combined. There are, however, further refinements which have been achieved by Forsell, Schwarz, Barclay, Akerlund, Baenisch, Chaoul, Milani and Fischer. All of these men have sought to so modify the original opaque-meal technic as to bring out details of the mucous membrane in various segments of the alimentary tract.

The present work relates somewhat briefly the historical development of the method and the apparatus with which the author's studies were carried out. Forsell, Barclay, Akerlund and Schwarz sought to make manual pressure against the anterior abdominal wall to bring out the details of the gastric rugae. Fischer sought to accomplish the same thing by the introduction of air, with the ingestion or injection of just a small amount of contrast material. Baensch, Chaoul and the present author have been leaders in the improved technic of securing mechanical firm pressure against the abdominal wall over the segment of the alimentary tract under study. Holzknecht sought to do this by means of a wooden distinctor, with which pressure was made by the hand while the film was being exposed. The later authors have accomplished this pressure by means of a balloon-belt apparatus, which was strapped around the patient. The balloon is then inflated until fluoroscopically it is seen that the proper amount of pressure is being made in the proper place, and then films are exposed. The resulting films reveal an astonishing wealth of detail regarding the mucous lining of the various portions of the alimentary tract, so that the method constitutes a distinctly valuable supplement to the current methods of barium diagnosis. The 193 illustrations in this book comprise a very fine library of reference roentgenograms, valuable to any diagnostician interested in gastroenterology.

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A CLINICAL STUDY OF THE ABDOMINAL CAVITY AND PERITONEUM

EDWARD M. LIVINGSTON, M.D.

PUBLISHED SERIALLY IN

The American Journal of Surgery

SIXTH INSTALLMENT

SECTION I. THE CAVITY (CONTINUED)

SECTION II. THE GASTROINTESTINAL TRACT

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A CLINICAL STUDY OF THE ABDOMINAL CAVITY AND PERITONEUM

Section 1. The Cavity

F. Pressure Variations within the Abdomen (*Continued*)*

5. Effects of Intra-abdominal Fat. Aside from the viscera with the liquids and gases they contain the chief content of the abdominal cavity is fat. Intra-abdominal fat, stored within mesenteries, ligaments and extraperitoneal spaces, acts as padding and serves to strengthen natural supports. Changes in the amount of this fat, as any change in the contents of the cavity, must be compensated for in some way. It has been shown, both clinically and experimentally, that when there is a marked loss of nitra-abdominal fat, there follows a compensatory atonic dilatation of hollow viscera, particularly of intestines.[92] Digestive and asthenic symptoms following an illness attended by a marked loss of weight may be accounted for in many instances by the loss of intra-abdominal fat and the secondary changes attendant upon this loss. The restoration of fat, because it strengthens mesenteric supports and causes a return of dilated gut to normal, is an important postoperative measure.

Preoperative Reduction in Weight with Large Ventral Hernias

A large umbilical or incisional hernia which has existed for a long time in a fat individual contains, in addition to other structures a deposit of fat. The amount of this fat tends to be in direct proportion to the size of the hernia. This overproduction of tissue may be so great as to make it difficult if not impossible to return all of the herniated mass to the abdominal cavity. Many complications may be avoided by securing in such patients a decided reduction in weight during

*Previous installments of this book appeared as follows: January issue, p. 193; February issue, p. 459; March issue, p. 693; April issue, p. 92; May issue, p. 1110.

an extended period of preoperative treatment. It has been said that if a weight reduction of 20 or more pounds can be obtained in patients having large ventral hernias, there will be little difficulty in securing complete reduction of the mass, and that postoperative complications will be appreciably less.[115] The high mortality following operation for large ventral hernias may be explained, at least in part, by the overcrowding of the cavity after abdominal closure.

6. Effects of Gravity. Gravity is most important among the factors which determine pressure variations within the abdomen. In the upright position the movable organs, generally speaking are approximately 3 cm. lower than in the prone position.[116] The organs lie in actual contact with one another, the upper being partially supported, as on a cushion, by the organs below. On account of the specific gravity of the viscera, the static pressure in the most dependent portions of the abdomen is considerable. The Fowler, knee-chest, and Trendelenburg positions and the elevation of the foot of the bed all illustrate common uses of gravity to alter intra-abdominal conditions.

An effect of gravity is noted in the spontaneous reduction of a hernia when the patient assumes a recumbent posture. The patient using a truss learns that it is of advantage to keep this device near the bed and to apply the truss before arising in the morning thereby being sure of a complete reduction and retention of the hernia. The visceroptotic patient may have learned the same regarding the application of his belt.

The Knee-Chest Position

That a change of posture results in a change in static pressures explains the condition known as "garrulitas vulvae." When the female patient assumes the knee-chest position, pressure is removed from the vaginal vaults as the movable organs of the pelvis and lower abdomen gravitate toward the diaphragm; and when the labia are separated air is admitted to the vagina; when the upright position is resumed a noisy expulsion of air takes place as the static pressure is restored in

the lower abdomen and the vaginal capacity is again reduced. Opening the vulva to admit air is an important, though frequently neglected part of the knee-chest exercise (see eponym:

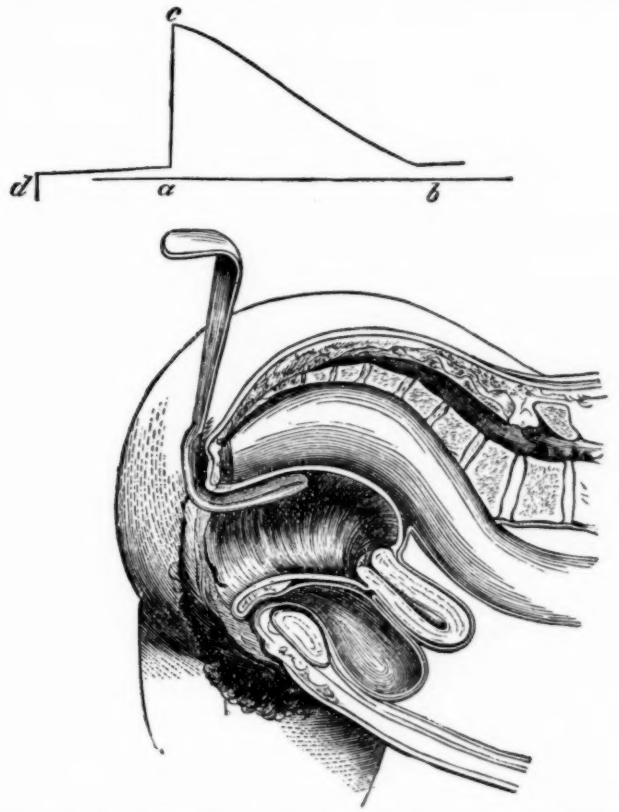

FIG. 85. The knee-chest posture. Note distention of vagina with air as labia are separated. (From Sims.)

Marion Sims);[117] so also is the taking of deep inspirations while in this position (combining the upward pull of gravity in the reverse static position with the upward pull or suction caused by subdiaphragmatic negativity). A woman assuming the knee-chest position may derive certain benefits therefrom, but will probably gain more through admitting air to the vagina and still more when adding deep breathing to the exercise (Fig. 85).

The relationship between local static pressures and general intra-abdominal pressures may be illustrated as follows: the

fundus of the uterus may rest forward upon the bladder or backward upon the rectum, causing in one instance bladder, and in the other, rectal-pressure; yet obviously the position of the uterus can have little bearing upon any pressure which may be present within the abdomen as a whole.

The Habitus Enteropticus

7. Effects of Individual Habitus. The habitus of the individual, particularly with regard to the shape of the lower thorax, and the erectness of the spine (a narrow chest and a forward curvature of the spine serving to diminish abdominal capacity) plays an important rôle in determining the character and distribution of abdominal pressures. Individuals with narrow, flat chests and sharp intercostal angles (habitus enteropticus) exhibit markedly different intra-abdominal arrangements from those of the square-chested type. Patients with congenital visceroptosis display a constitutional weakness in supporting tissue.[118] This is noted by the malposition of one or many organs, the partial or complete failure of normal embryonic rotations and fusions in the gastrointestinal canal and the continuous display of subjective abdominal symptoms of a distressing nature. There seems also to be an excessive tendency to the formation of intraperitoneal adhesions. The small capacity of the upper abdomen crowds the viscera to abnormally low levels and produces lengthening and traction upon intra-abdominal supports, thus markedly accentuating such symptoms as are caused by any downward displacement of the organs. The abdomen presents an s shape, being flat or retracted above and protuberant below (Fig. 86).

Subjective Symptoms from Visceroptosis

Subjective symptoms are due chiefly to stretching and irritation of the splanchnic nerves and vagi together with torsions and pressures upon local blood vessels. These range from the dull gnawing feeling within the epigastrium and the slight faintness or giddiness experienced after going for a long period without food, to the severe grades of gastrointestinal, cardiovascular, pulmonary, nervous and nutritional disturbances, noted with marked visceroptosis, constituting the sympathetic imbalance of a true Glenard's disease.[118]

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Visceroptosis represents one of the most important of surgical studies. Though common, this condition all too frequently remains unrecognized and the patient may pass through many

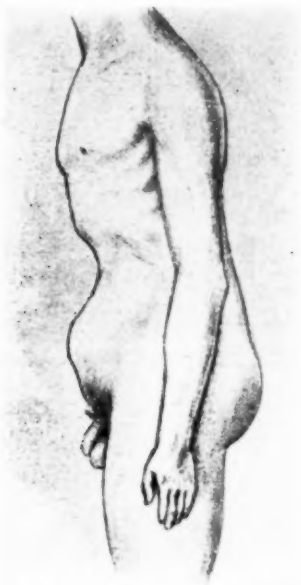

FIG. 86. Contour of the abdomen with marked visceroptosis. (From French.)

hands, both medical and surgical, before recognition is taken of the stigmata present. Patients having the acquired form of visceroptosis complain chiefly of gastrointestinal symptoms while those with the congenital type of this condition are, as a rule, classed as neurasthenics. The symptoms, which are most varied, may simulate several abdominal diseases and lead to operative intervention which usually proves disappointing as to end-results.[120] A report has been given concerning 1000 such patients, averaging two and one-half operations each, but presenting a chain of baffling symptoms and syndromes which were little influenced, unless exaggerated by operation. It is not implied that with visceroptosis operations are always contraindicated but they should certainly be undertaken only after due deliberation, and the results should be interpreted in terms of the developmental stigmata which underlie the condition.

Methods for Visceral Elevation

The subjective symptoms due to displacement of viscera are, as a rule, promptly relieved by support, if this adequately replaces the viscera. Relief is less certain with the congenital than with the acquired type of visceroptosis, because in the former, the shape of thorax and abdomen makes complete replacement difficult or impossible, and the nervous symptom-complex is of such long standing as to be more or less fixed in degree. Measures aimed at a replacement of the organs include[118–120] sleeping with elevation of the foot of the bed; regular assumption of the knee-chest, Trendelenburg, or some other reverse-static posture; prolonged rest in bed with the administration of a fattening diet calculated to shorten ligamentous and mesenteric supports; the use of mechanical binders and straps; the employment of special athletic exercises; and occasionally, operative shortening or rearrangement of abdominal supports. The extent and type of treatment naturally will vary with the severity of symptoms. Treatment, of whatever type, tends to prove quite ineffective unless the viscera are actually elevated and are so supported as to remove downward traction; but when this is efficiently accomplished, prompt relief may be anticipated. The natural support given the organs by the pregnant uterus often grants the visceroptotic woman a period of relief from symptoms and demonstrates the value of adequate elevation and visceral replacement.

Recapitulation Concerning Intra-abdominal Pressures. Pressure changes within the abdomen are complex. To assume a close analogy as to pressure conditions between abdomen and thorax must lead to errors. The thorax has a special anatomical composition (rigid walls) to allow for a special physiological activity (respiration) which is entirely dependent upon a reasonably constant relationship between the pressure within and that of the atmosphere. Intrapleural pressure is little effected by gravity, by variations in fat content or size of viscera, or by many other factors which have been seen to play so important a rôle in determining pressures

within the peritoneal cavity. Neither abdominal structure nor abdominal function implies a uniform pressure throughout the peritoneal cavity nor any constant pressure range within this potential space.

The meaning of the term intra-abdominal pressure is necessarily somewhat vague. It is not incorrect to refer to increased intra-abdominal pressure where, by a sudden contraction of the muscular walls, any previously existing pressures are grossly increased; or again, where there is a bulging of the walls from some obvious, generalized and considerable elevation of pressure. An analogous generalized and marked pressure-decrease may be spoken of as a reduction in intra-abdominal pressure. But it must be borne in mind that pressure conditions within the abdomen are a combination of intraperitoneal pressures, static pressures, and intravisceral pressures; and that changes and adjustments are continually being made.

Basically the mean intra-abdominal pressure tends to be equal to that of the atmosphere, for the abdomen, a non-rigid sac, is directly subject to atmospheric pressure and its walls tend passively to bulge outward when the pressure within exceeds that of the atmosphere (ascites; paralytic ileus) or to passively move inward when the pressure within is below that of the atmosphere (dehydration; cachexia). But the abdomen is a muscular sac capable, by voluntary contraction of its walls, of suddenly increasing any pressure within (cough) and by continued involuntary contraction (reflex rigidity) of maintaining such increased pressure; the muscles also, through vital reflex phenomena, may relax and thus decrease intra-abdominal pressures (abdominoenteric reflex). Because of the location of the abdomen, separated from the pleural cavities only by a thin musculofibrous membrane, the upper portions of the peritoneal cavity display a slight reflected negativity (subatmospheric pressure or tension phenomenon) showing the presence of an upward pulling force. Because of location, also, the intra-abdominal pressures show variations

with the phases of the respiratory cycle; and these, in turn, are influenced by the habitus of the individual and his type of breathing, the relative negativity in the upper peritoneal cavity being greatest when breathing is of the thoracic type and during the inspiratory phase of the respiratory cycle, and least when breathing is of the abdominal type. Abdominal pressures, again, vary continuously with changes in intravisceral pressures (those within individual organs). Any change in pressure within one organ is, of necessity, compensated for by changes in other abdominal viscera or in the abdominal walls. Gravity also is a most important controlling factor in determining pressure variations within the abdomen.

When the surgeon means specifically to refer to intravisceral or intraperitoneal pressures, or to those due to gravity (static pressures) he should, when possible, so state. The success of diagnostic methods and therapeutic measures which are in any way dependent upon pressure variations within the abdomen will tend to bear a direct relationship to the accuracy with which the true causes of the pressure variations are interpreted.

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Section II

THE GASTROINTESTINAL TRACT

THE abdomen has thus far been considered simply as an empty space. It has first been investigated as a cavity devoid of viscera and emphasis has been placed upon the interrelationships between the abdomen and other parts of the body. That perspective has been sought which would ensure the truest understanding of the cavity as a whole. And now, by a closer approach, attention will be directed to the abdominal contents and intra-abdominal organs will be individually studied.

As attention is thus withdrawn from the abdominal walls interest at once centers upon the gastrointestinal tract. The enteric canal with its voluminous intestinal loops and massive glandular outgrowths practically fills the abdomen (Fig. 87). It has a length equal to from five to six times that of the body itself. Alike by fact and by derivation of terms the intestines constitute the "inwards" or the "insides" of the belly (L. *intus*, within) (entrails, L. *interaneus*, internal, inward, interior). As is readily apparent the space within the abdomen is given over almost entirely to processes of alimentation (L. *alimentum*, food, nourishment). This extreme length of the alimentary canal represents an adaptation to function. Digestion (L. *digerere*, to dissolve or divide) is in part chemical and in part mechanical and this necessitates the provision of a large lymphatic and vascular absorptive area and of adequate storage space both for foods and waste products. Occupying the major portion of the abdominal cavity the gastrointestinal tract becomes a major consideration in any review of this space.

It seems quite generally agreed that there is but one logical point at which to begin a broad investigation of this tract, namely with a study of its origin. Embryology has proved the most reliable guide in explaining adult conditions.

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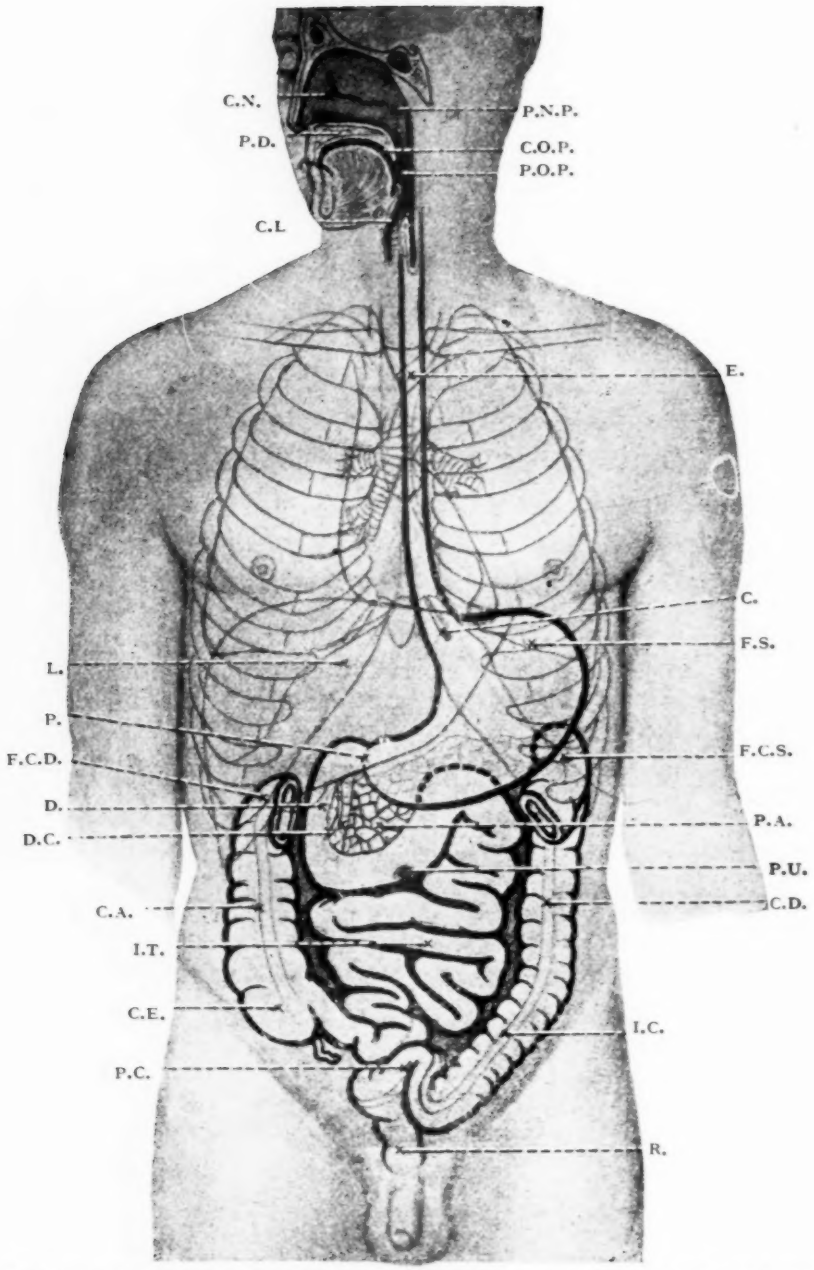

FIG. 87. Note that the enteric canal with its voluminous intestinal coils and massive glandular outgrowths practically fills the abdomen. (From Cunningham.)

C.N., Cavum nasi. P.N.P., Pars nasalis pharyngis. P.D., Palatum durum. C.O.P., Cavum oris proprium. P.O.P., Pars oralis pharyngis. C.L., Cavum laryngis. E., Esophagus. C, Cardia. F.S., Fundus of stomach. L, Liver. P, Pylorus. F.C.S., Flexura coli sinistra. F.C.D., Flexura coli dextra. D, Duodenum. D.C., Ductus choledochus. P. A., Pancreas. P.U., Position of umbilicus. C.A., Colon ascendens. C.D., Colon descendens. I.T., Intestinum tenue. C.E., Cecum. I.C., Iliac colon. P.C., Pelvic colon. R, Rectum.

Anatomical and physiological considerations which might otherwise remain almost inexplicable are rendered relatively simple through a study of the development of the digestive tube. Many observations made at the bedside and many manouvers suggested for the surgical amphitheater are rendered clearly intelligible only through insight into the series of changes which take place during embryonic life.

In the present post-graduate review, therefore, no short-cut will be attempted from this somewhat laborious method of tracing step by step succeeding developmental alterations, for the inevitable reward is a more clear understanding of both diagnostic and operative matters. A clinical review, it might be pointed out, has as its immediate aim only the study of points which are essentially practical. But to fully appreciate bedside observations some fairly accurate knowledge must first be had of the mechanism by which signs and symptoms are initiated. Correct diagnostic conclusions are necessarily dependent upon a correct interpretation of the diagnostic data. In succeeding pages, then, there will be combined and correlated in the most intimate fashion topics which are elemental and basic with those which are purely clinical in character and the attempt will be made, not only to discuss the clinical observations themselves, but in addition to take up the manner in which they may be produced and to suggest ways in which they may be accounted for.

A. BASIC EMBRYOLOGICAL CONSIDERATIONS

The abdominal portion of the body consists fundamentally of a tube suspended within a tube (Fig. 88). The outer of these is the abdominal wall, the inner the gastrointestinal tract. The external surface of the body is derived from ectoderm and is protective and "relational" in function, while the innermost surface (i.e., the inner lining of the inner tube, which in actual square surface more than equals the area of the skin itself) is derived from endoderm and represents the nutritional tissue of the body. Between these tubes is that

potential space, its walls derived from mesoderm, which is variously known as the abdominal portion of the body cavity, the adult celomic cavity, the extravisceral cavity,

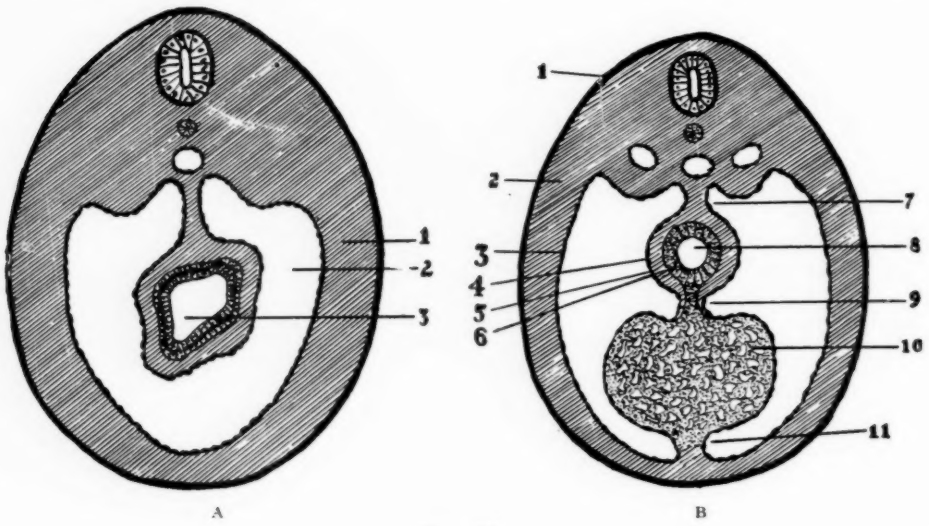

FIG. 88.

A. The abdominal portion of the body consists fundamentally of a tube suspended within a tube; between these tubes lies the peritoneal cavity.

1. Outer tube or body wall. 2. Peritoneal cavity. 3. Inner tube or bowel.

B. An earlier stage of development (cross section above level of umbilicus).

1. Ectoderm. 2. Somatic mesoderm. 3. Endothelial lining of peritoneal cavity (parietal peritoneum). 4. Visceral peritoneum. 5. Splanchnic mesoderm. 6. Endodermal lining of bowel. 7. Primitive dorsal mesentery. 8. Lumen of bowel. 9. Primitive ventral mesentery. 10. Liver. 11. Future falciform ligament of liver.

or the peritoneal cavity. The study of these arrangements and of the clinical significance of the three primary germ layers, involves a consideration of four topics:

1. Origin of the gastrointestinal tract.
2. Origin of the peritoneal cavity.
3. Distribution of the adult gastrointestinal tract.
4. Final attachments of the gastrointestinal tract.

In reviewing the peculiar manner in which viscera and body walls are connected, the operating surgeon is chiefly interested in vascular and peritoneal or membranous connections; while for the surgical diagnostitian the dominant interest is in nerve connections.

1. Origin of the Gastrointestinal Tract. It will be recalled that the enteric canal (inner lining of the gastrointestinal tract) develops from the yolk sac.[121] It is formed from this structure as the embryonic streak or rudiment bends forward at its cephalic and caudal extremities while creating the anterior abdominal wall (Fig. 89 B). At first the enteric canal is blind at its cephalic and caudal ends while the middle part of its ventral wall is continuous with the wall of the yolk sac (endodermal lining) into which the archenteron (G. first or primitive intestine) is open for about one-third of its length (Fig. 89 C). The portion of the tract thus open into the yolk sac is termed the midgut; that cephalad to this opening, the foregut; and that caudad, the hindgut. The opening into the yolk sac gradually narrows as the anterior abdominal walls close in, but the archenteron retains a connection with the diminishing yolk sac (now a mere yolk vesicle) by a slender tube of smaller and smaller caliber, the yolk stalk, or vitello-intestinal duct (vitelline, L. yolk) (Fig. 89 D). With the degeneration of the yolk sac into a yolk vesicle this vitello-intestinal duct exerts a traction upon the intestine at its point of attachment and thus draws a loop of small bowel out of the body cavity into the umbilical cord. This intestinal loop, with the duct at its apex, consists of two limbs (Fig. 89 E). The first of these has been variously termed the proximal, descending, leaving or efferent limb; the second is known as the distal, ascending, returning or afferent limb. This protrusion of bowel into umbilical cord is known as the "embryonal umbilical herniation." The loop begins to enter the cord in the 10 mm. embryo (three weeks) and the degree of herniation increases until the tenth week of intrauterine life. Normally after this period the bowel returns to the abdomen (explained below). The umbilicus is the last point in the abdominal walls to close and the navel or omphalos represents the central point of the anterior body wall (navel, L. center; *omphalos*, G. a central object). In the adult the only trace of the early connection between intestinal canal and

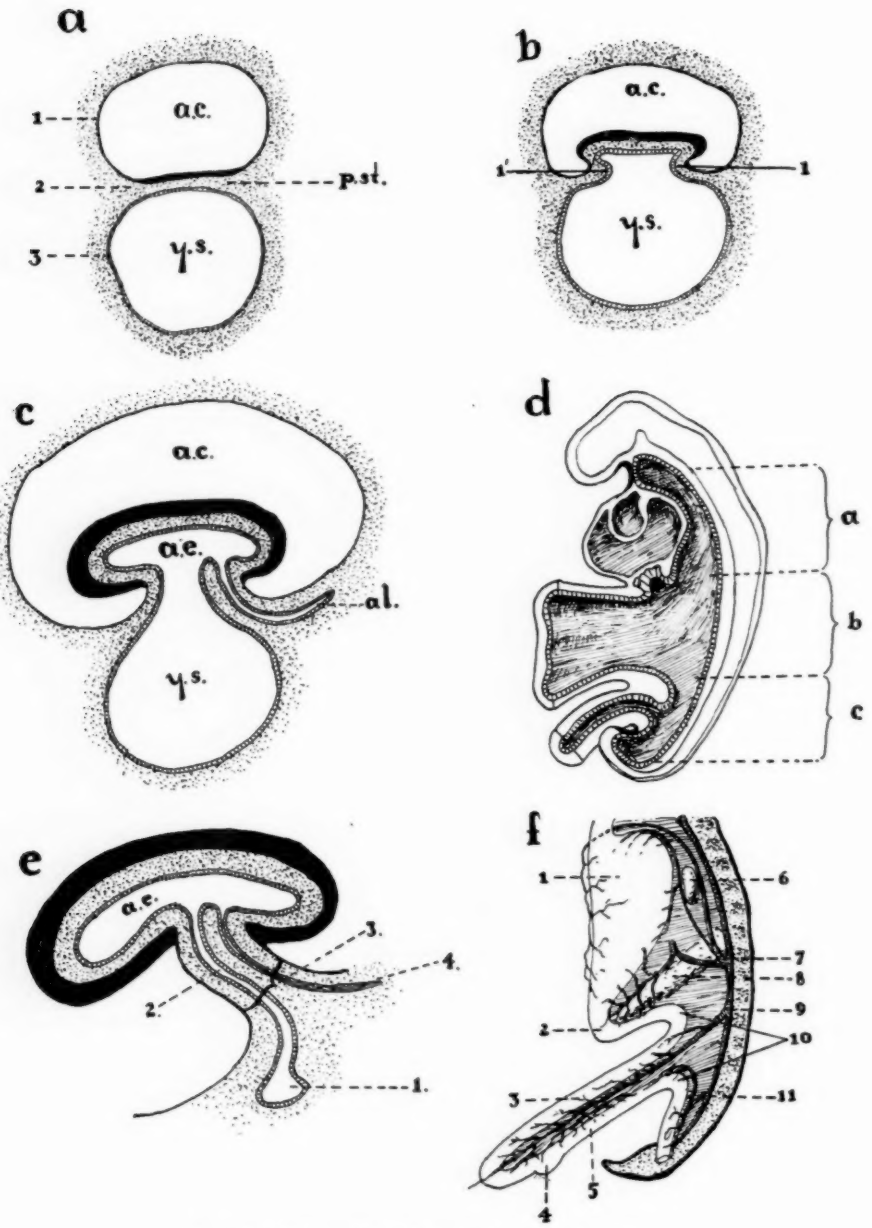

FIG. 89. Formation of gastrointestinal tract.

A. The earliest embryonic rudiment or primitive streak (p. st.), which lies between the amnionic cavity (a.c.) and the vitelline cavity or yolk sac (y.s.).

1. Ectoderm. 2. Mesoderm. 3. Endoderm.

B. As the embryo bends forward at its cephalic (1′) and caudal extremities (1) the yolk sac becomes constricted and the first evidence of an alimentary canal (embryonic endoderm) appears.

umbilicus is the rare (2 per cent) persistence of a diverticulum or rudiment of a diverticulum, uniting the lower portion of the ileum with the navel (Meckel's diverticulum occurs more or less distinctly in one person in fifty). The visualization of the origin of the enteric canal is aided through the use of accompanying figures.

Abnormalities at the Navel

This brief statement of developmental facts concerns matters which have considerable practical significance. Mention has previously been made of such clinical points as the occurrence of fistulae, cysts and endodermal neoplasms at the navel, the explanation of which depends upon an understanding of the origin of the enteric canal; the persistence at birth within the umbilical cord of a loop of bowel, necessitating care to strip the cord backward to the body wall between the thumb and forefingers before applying clamps or ligatures, has been taken up; the incidence of congenital umbilical hernias has been mentioned, together with the fact that their spontaneous cure within the first two years of life is the rule, due to the inward traction of the umbilical ligaments (obliterated urachus; obliterated hypogastric arteries).

Congenital Diverticula of Ileum

Diverticula due to the complete or partial persistence of the omphaloenteric or vitelline duct vary widely as to their point of origin from bowel, their site of attachment within the peritoneal cavity, and as to size and shape. It is very often stated that a Meckel's diverticulum, if present, will be found "within 12 to 14 inches of the ileocecal valve,"[122,123] but the point of origin as a rule is at a considerably greater distance

Continued from p. 244.

C. The alimentary endodermal tube further constricted from the yolk sac.

a.e., Alimentary endoderm. y.s., Yolk sac. al., Allantois. a.c., Amnionic cavity.

D. The alimentary tract remains open in its mid-third into the diminishing yolk sac.

a. Foregut (blind tube). b. Midgut (open anteriorly). c. Hindgut (blind tube).

E. Alimentary canal, a closed tube except for its communication with the yolk vesicle through the vitello-enteric duct and into the allantois.

a.e. Alimentary canal (endoderm). 1. Yolk vesicle. 2. Vitello-enteric. duct. 3. Umbilical cord. 4. Allantois.

F. The primary loop of the gastrointestinal tract.

1. Stomach. 2. Duodenum. 3. Descending limb of intestinal loop. 4. Cecal bud. 5. Ascending limb of loop. 6. Spleen. 7. Celiac axis. 8. Pancreas. 9. Superior mesenteric artery. 10. Duodenocolic isthmus or base of loop. 11. Inferior mesenteric artery.

from the large bowel. Meckel himself simply gave the place of origin as "in the small intestine and nearer its lower than its upper extremity"[124]; he stated that it "varies to a considerable extent." Huntington found by actual measurement in 22 instances of this anomaly that the average point of attachment to bowel was located 107 cm. or 3 ft. 6 in. from the ileocecal valve.[125] The obvious practical significance of this fact is that when operating for intestinal obstruction of unknown cause, search for a congenital diverticulum of the ileum as a possible cause, should extend over a wider area than the terminal 1½ feet of ileum. At least the terminal 3½ feet of ileum must be examined to discover the average case and the presence of a diverticulum cannot be positively ruled out unless over 9 ft. of terminal ileum are inspected.

A Description of the Diverticulum of Meckel

A Meckel's diverticulum is always single. The diverticulum arises from the free margin of bowel (at a point opposite to the mesenteric attachment) and usually passes away from the ileum at a complete or right angle (Fig. 90 A). This congenital anomaly contains all of the coats of the bowel wall, a circumstance which serves to distinguish it from false or acquired diverticula. The diverticulum at its point of origin may have a caliber equal to that of the ileum itself, or it may be smaller, even being a cord with the finest of central apertures (Fig. 90 B). A Meckel's process is always broader at its point of origin than at any other point. It frequently tapers rapidly as it leaves the ileum, although the extreme tip may be somewhat clubbed or bulbous, due to a herniation of the mucus through the muscular coat (Fig. 90 C). At times the diverticulum is a mere pouch or nipple-like process in the lower portion of the ileum. The diverticulum may extend directly to the umbilicus, usually as a cord but rarely as an open duct; it may attach internally to the midline at a varying distance from the umbilicus; at times its extremity is attached to intra-abdominal structures, notably the mesentery of the bowel or the bowel wall (Fig. 90 D); in still other instances, and quite commonly the terminal extremity of the diverticulum

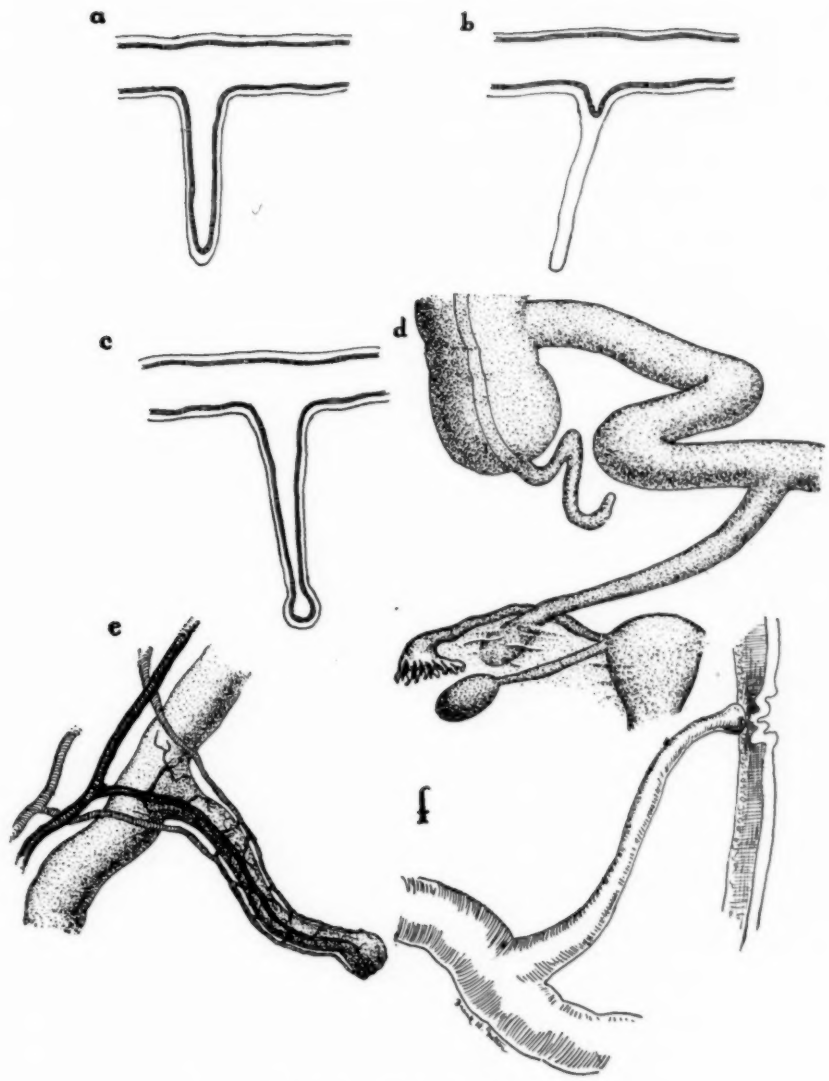

FIG. 90. Varieties of Meckel's diverticulum.

A. Diverticulum leaves bowel at free border (opposite mesenteric attachment) at right angles to long axis of bowel.

B. At times the process is little more than a fibrous cord.

C. The diverticulum is always largest at its point of origin from bowel, but sometimes has a bulbous tip.

D. Tip of diverticulum has acquired a secondary attachment (in this case to broad ligament).

E. Showing vessels (these are called vitello-mesenteric vessels) which may accompany the diverticulum. Note how the vessels pass over and may constrict bowel wall.

F. Diverticulum passing to umbilicus; small endodermal cyst at anterior abdominal wall.

lies free and unattached within the peritoneal cavity, the process having an average length of 3 inches.

Persistent Omphalo-mesenteric Vessels

Vessels may accompany the diverticle, whatever its course. These may consist of an artery and two veins.[124] They arise from branches of the superior mesenteric vessels, cross over the ileum opposite their points of origin, and traverse the walls of the congenital pouch (Fig. 90 E). These vessels may themselves constrict the bowel. They originally had the function of nourishing the yolk sac and are therefore called the omphalo-mesenteric vessels. In the rarest of instances the arteries or fibrous cords representing them may pass from their mesenteric origin to the region of the umbilicus even when there is no congenital diverticulum of the bowel.

Comparison of True with False Diverticula of the Ileum

In contrast to the diverticulum above described, acquired or false diverticula of the ileum are almost without exception multiple.[126] They arise along the attachment of the mesentery to bowel wall, not at the free bowel border (Fig. 91). They are short; they are polypoid or clubbed, and at their point of origin are narrower than at other parts. Although subject to secondary inflammation they do not cause mechanical obstruction of bowel. They occur in old people, rarely in the young. These multiple acquired outgrowths are caused by protrusion or herniation of mucous lining through the muscle coat; they never, as does a Meckel's diverticulum, contain a covering of muscle. False diverticula, then, differ from a Meckel's process in all essential points, in structure, number, shape, position, size, age incidence and potentialities.

The omphaloenteric duct or persistent omphalo-mesenteric vessels give rise to intestinal obstruction in 4 ways: (1) by bowel strangulation under a fibrous cord or diverticulum; (2) by loops or nooses of the diverticulum or a cord; (3) by those remarkable formations known as diverticular knots, and (4) by the passage of the diverticulum into a hernial sac (Fig. 92).

The first method is by far the most common (outnumbers the other causes by over three to one). Obstruction rarely occurs when the attachment of the diverticulum is to the

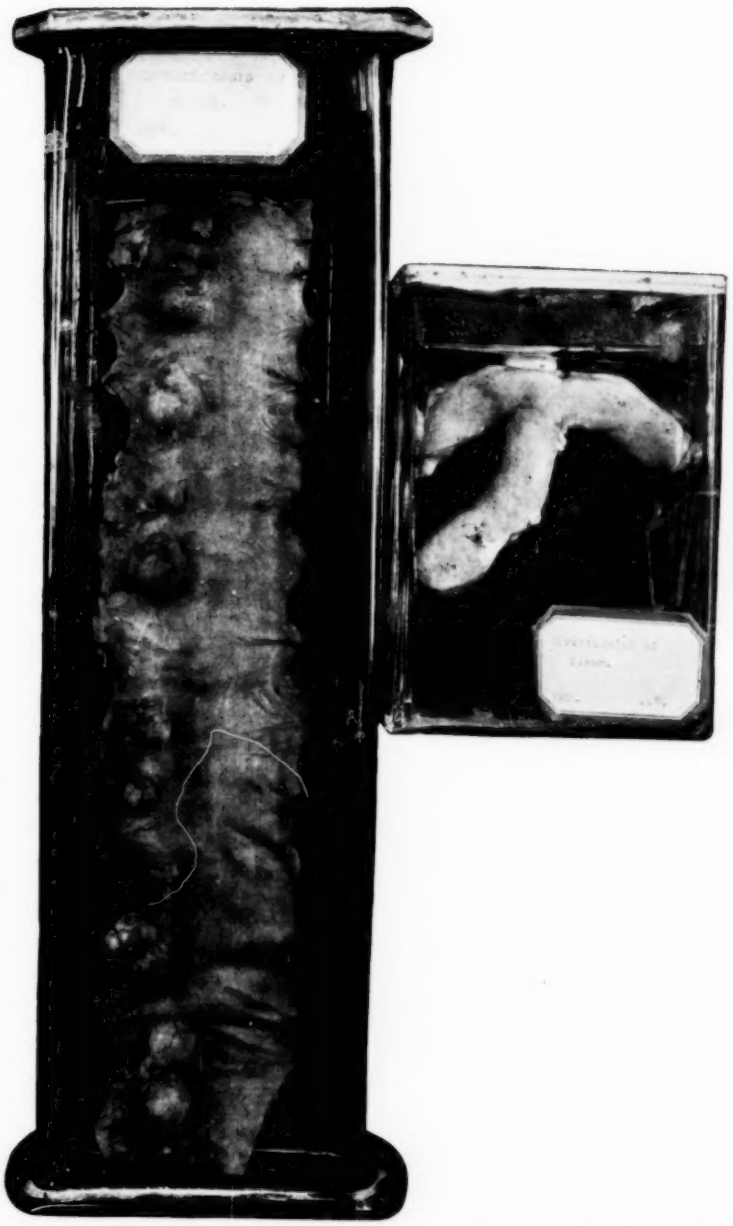

FIG. 91. Differentiating a congenital diverticulum (Meckel's) from acquired or false diverticula. The former is single, contains all coats of bowel, arises at free border of bowel, has its largest caliber at its point of origin and is 3 inches in length. The false diverticula are multiple, short, pedunculated, contain no muscular coats, and arise along the attachment of the mesentery to bowel, not opposite to free bowel margin. (From 3rd Surg. Div., Bellevue Hosp.)

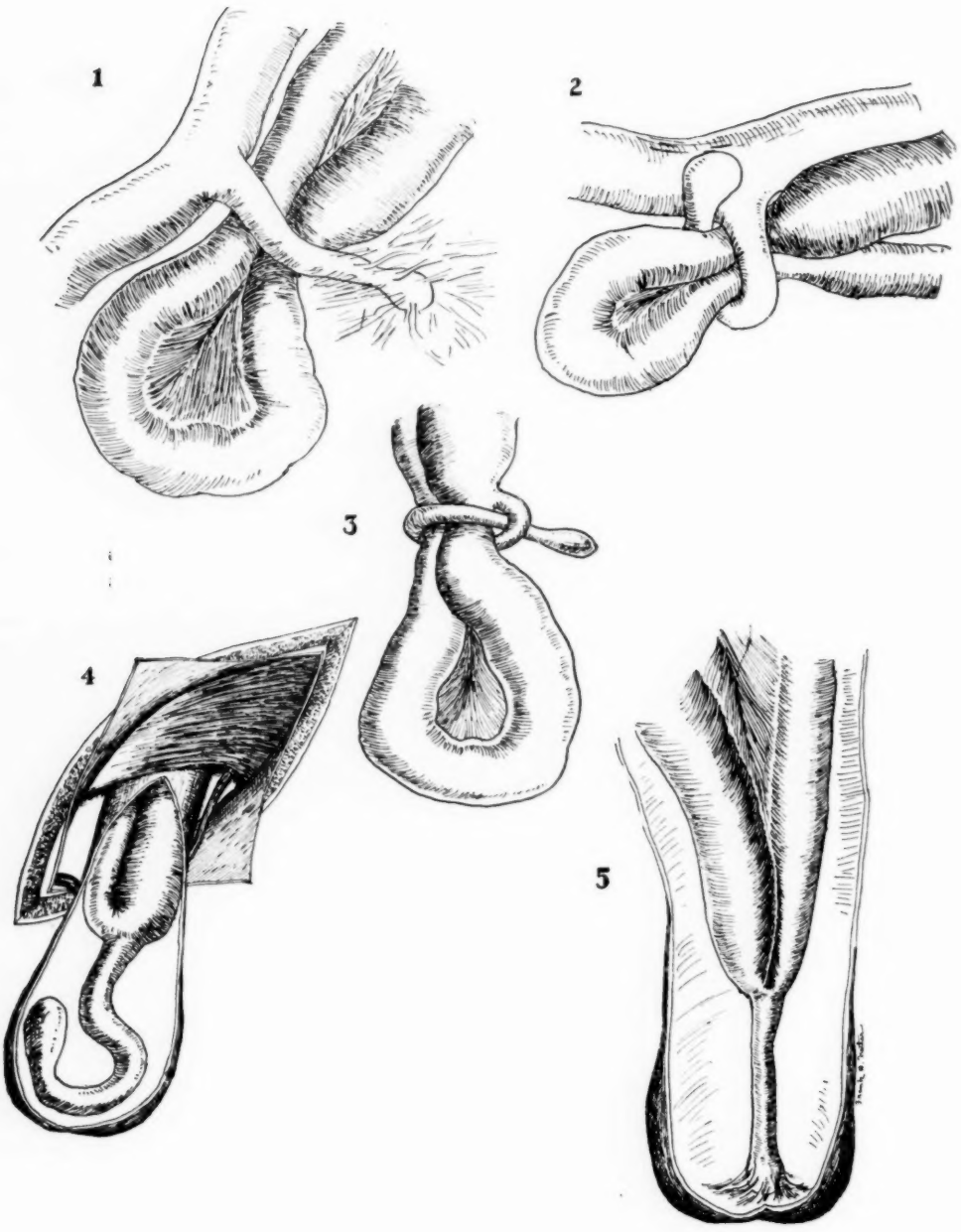

FIG. 92. Intestinal obstruction from congenital diverticula (Meckel's).

1. Obstruction beneath diverticular band. 2. Obstruction by a diverticular noose. 3. Obstruction by a diverticular knot. 4. Obstruction of diverticulum within a hernial sac. 5. Obstruction from angulation of ileal loop by diverticular traction.

Varieties of Intestinal Obstruction from Persistent Omphalo-enteric Ducts

umbilical region, but commonly occurs when there is a secondary attachment to mesentery or bowel. These points of secondary attachment are often determined by intraperitoneal inflammations and have been reported at the site of old inguinal hernias, sclerotic mesenteric glands, or to inflammations about ovaries, tubes or appendices. When the diverticulum is short only a small knuckle of bowel is capable of engaging beneath the resultant loop, but when the loop is long a large portion of bowel may become either incarcerated or strangulated. The diverticulum sometimes forms a loop or a compound loop into which bowel becomes engaged and subsequently ensnared as a later change occurs in the lines of traction within the belly. Intestinal obstruction from knots is explained graphically in accompanying figures. A bulbous enlargement of the tip of the diverticulum, as previously referred to, plays an important rôle in the formation of these peculiar knots, for once the knob-like process has become engaged through an aperture it does not again readily become disengaged.

Site of Pain From Gastro-intestinal Affections

The study of embryology aids not only in an understanding of diverticula but also in the interpretation of symptoms produced by gastrointestinal disorders. It proves particularly valuable in explaining the localization of pains due to alimentary tract affections. The six-weeks' embryo with its primary loop of bowel and its simple distribution to this loop of vessels and nerves, furnishes a picture which makes readily intelligible the distribution of such pains to the midline of the anterior abdominal wall. To determine the exact sites to which the patient refers his pains is a matter of the utmost importance in the diagnosis of surgical disorders within digestive organs. As early as 1892 MacKenzie published results of a careful inquiry into this pain-reference as described by 320 patients suffering from affections of stomach.[127] He found the pain in 95 per cent of cases to be localized within the epigastrium. Subsequently, after keeping records of several thousands of cases, he reached the conclusion that the patient does not

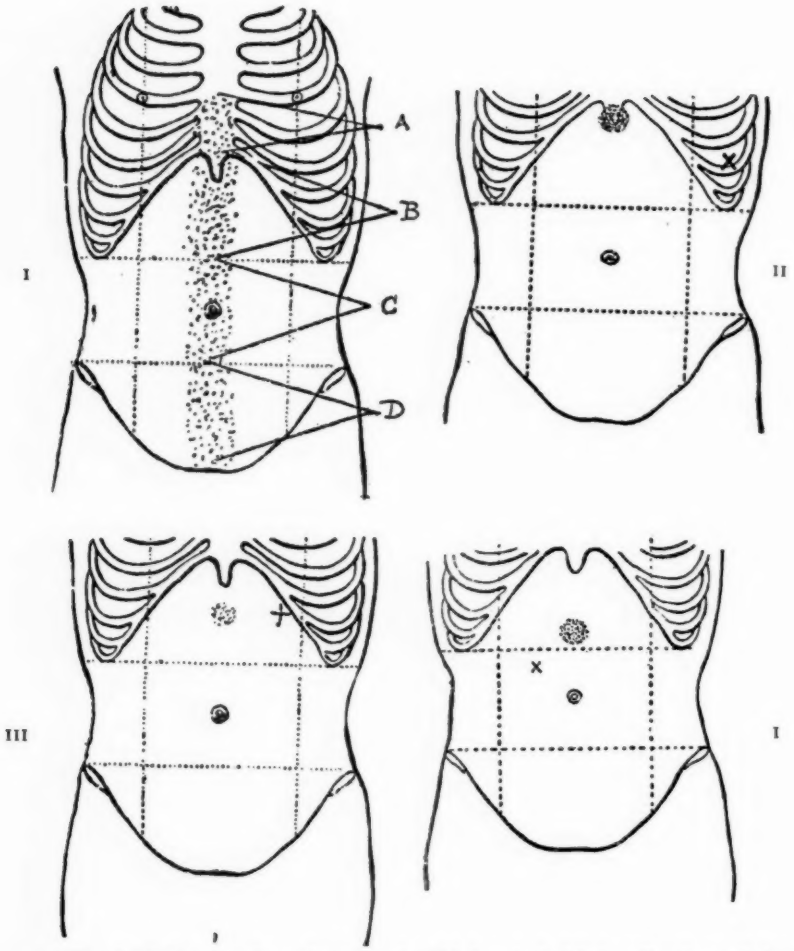

FIG. 93. I. The shaded parts show the areas in which pain is felt in affections of the digestive tube. (From Mackenzie.)

A. Area in which pain is felt in affections of the esophagus. B. Area in which pain is felt in affections of the stomach. C. Area in which pain is felt in affections of the small intestine. D. Area in which pain is felt in affections of the large intestine.

II. The shaded area shows site of pain, x corresponds to the position of the ulcer in the stomach as found at the post-mortem examination.

III. The shaded area shows the site of pain, and the position of the gastric ulcer as found at operation.

IV. The shaded area shows the site of pain, x the site occupied by the ulcer at the pyloric orifice of the stomach as found at the post-mortem examination.

actually feel pain within the viscera themselves but refers it to the abdominal walls and to the area of distribution of sensory nerves of the cerebrospinal system. And according to the observations of Mackenzie the pain from digestive disturbances is interpreted by the patient as arising at the periphery of this sensory nerve distribution, that is, approximately at the midline of the body anteriorly. He stated "from the top of the oesophagus to the anus there is, in the vast majority of cases, a limitation of the distribution of the pain to an area extending down the centre of the body from the middle of the sternum to the symphysis pubes."[127] (Fig. 93.) Thus, when the inner lining of the oesophagus is irritated by a too hot drink, pain is always referred to a region over the lower part of the sternum; organic lesions of stomach, whether located near cardia or pylorus, give rise to pain which is referred to the epigastrium; such conditions of small bowel as strangulation of an intestinal loop yield pain which is felt in the neighborhood of the umbilicus; and again, affections of large bowel (volvulus of sigmoid, diverticulitis, stricture) when these cause pain, cause it within the hypogastrium.

Pain Reference to the Periphery

There are several ways of demonstrating that the pain is not actually felt "within" esophagus, stomach, or bowel or "inside of the belly" as the patient may himself believe and state. The following facts well illustrate the reference of the pain to the tissues of the body walls; gastric pain is epigastric in location although the stomach itself may lie at a lower level or even extend into the hypogastrium or pelvis and the epigastric pain remains constant in location although the stomach is known to shift about during periods of functional activity. Again, while the intestinal loops twist and coil throughout the entire infracolic division of the abdominal cavity and are quite largely hypogastric and pelvic in location, small bowel pain without accompanying peritonitis is referred with uniformity to the area about the umbilicus.

Mackenzie's original illustration of the areas in which pain is felt in affections of the digestive tube is reproduced in

The Three Equidistant Pain Zones of the Midline

Figure 93. Considering the anatomical distribution of the various components of the gastrointestinal tract, the nature of this reference of pain to the midline of the body is astonish-

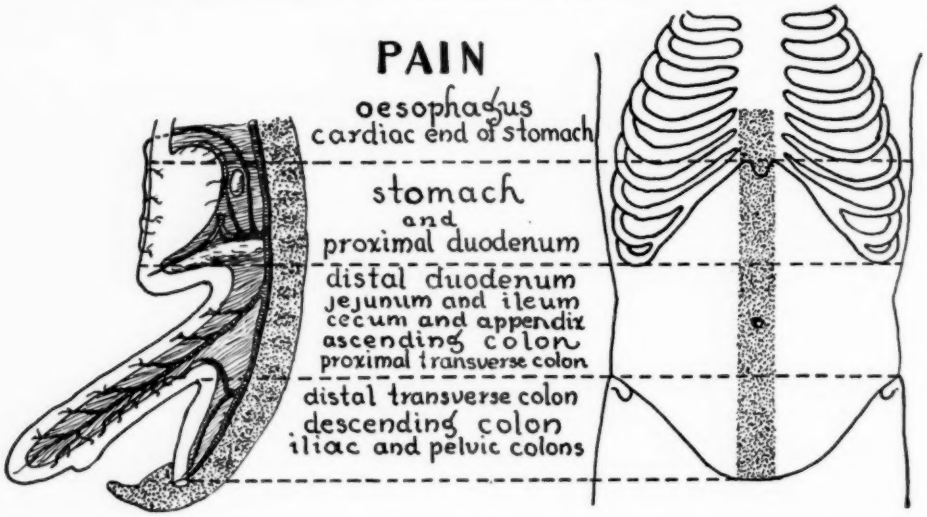

FIG. 94. Study of reference of gastrointestinal tract pains to three equidistant zones in midline of anterior abdominal wall.

ing. Why does it occur that affections of stomach and duodenum give rise to pain which is felt over some 6 in. of the midline while affections of small intestine, within any part of its many feet of tortuous course, have a body-wall zone of no greater size (the 5 6 in. about the umbilicus)? What accounts for this inequality of length between the areas of pain-reference and of the bowel divisions themselves? Why is it, also, that large bowel pain is situated at a lower point than small bowel pain, when anatomically the colon describes a loop about the periphery of the abdomen and has right and left flexures which reach to the hypochondriac regions, while the terminal ileum is situated at such a low point in the abdomen or even within pelvis? It is when the adult surface map for pain is juxtaposed to a picture of the six-weeks' embryo that the situation becomes easily clarified (Fig. 94). It may be noted that the upper third of the abdominal portion of the archenteron gives rise to the stomach and proximal duodenum;

that the mid-third gives rise to terminal duodenum, all remaining parts of small intestine, to cecum, appendix, ascending colon, and to the proximal portion of the transverse colon; that the distal third of the primitive bowel gives rise to the remainder of the colon. Since the nerve tissue is being distributed at this period of development (see invasion of ectodermal tissue into the abdominal cavity) and since the central nervous connections are established at this time, it follows that all subsequent twists, elongations or other changes occurring in individual portions of the tract have but little effect upon adult reflex pathways. The relationships of the three primary divisions of the archenteron to the three equi-distant zones of the midline of the anterior abdominal wall remain fixed and constant.

Gastric Lesions vs. Epigastric Distress

But while stomach disorders produce, with great regularity, pain which is epigastric in situation, it by no means follows that epigastric distress always signifies some gastric lesion. The stomach has been referred to as "the spokesman of the abdomen"[128] and roughly only about one-third of stomach symptoms are due to actual gastric lesions. Another third are caused by extragastric diseases existing within the abdomen or pelvis, as for example cholelithiasis, chronic appendicitis, tuberculous peritonitis, ovarian cyst, salpingitis. The remaining third of gastric symptoms are due to conditions not even located within the abdomen but existing entirely outside of this cavity as, a basilar meningitis, brain tumor, pulmonary tuberculosis, or exostosis associated with a lower thoracic vertebra. (The mechanisms by which these facts are explainable are discussed elsewhere.) Lesions at almost any point in the body may cause gastric symptoms through a direct or indirect involvement of gastric vessels and nerves. A list of such conditions, would necessarily be long and include general headings as endocrine disturbances, psychic disturbances, drug poisonings, abdominal allergies, etc. The ability of the diagnostician to arrive at a correct diagnosis will be limited unless he is thoroughly familiar with the many medical and surgical

conditions represented by the extensive list of actual causes. Given a patient, then, complaining of gastric symptoms, a complete study of all phases of the case is an obvious necessity

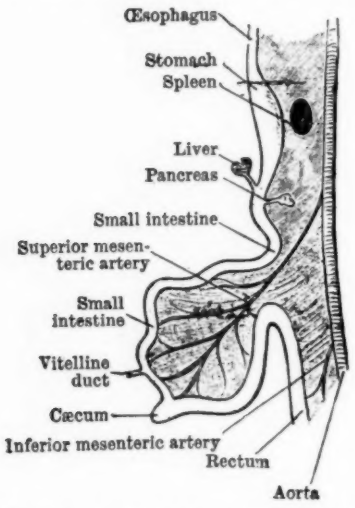

FIG. 95. The duct system of the liver arises as an outgrowth of the developing duodenum. (From Waterston. Cunningham's Anatomy.)

in arriving at a correct diagnosis. This means not only a thorough examination of all parts of the abdomen and pelvis but also of head, neck, thorax, extremities and of every region of the body; it involves not only a careful history but adequate laboratory tests as well. The abdominal spokesman simply tells of the presence of trouble; it remains for the diagnostician to definitely identify the organ or tissues diseased.

Chronic Dyspepsia and Gall Bladder Disease

There are, nevertheless, certain clinical rules and guides in dealing with epigastric distress which may be relied upon to a considerable extent as indicating, at the first examination of the patient, the true diagnosis. Perhaps the most common extragastric cause for epigastric distress is some affection of the biliary adnexa. The possibility of gallstone disease should always be borne in mind in any case of chronic indigestion. The bile ducts (endodermal portion) arise as an outgrowth of the developing duodenum (Fig. 95) and it might therefore be anticipated that if they produce, as do remaining portions

of the enteral tube, a reference of pain to the midline of the anterior abdominal wall, the site of this referred pain would either be some region between the surface zones for gastric and small intestinal pain or would coincide with that for duodenum. In other words, there are theoretical reasons why the pain with biliary tract disorders should be felt within the epigastrium. And the study of clinical records shows that in 20 to 40 per cent of cases of cholelithiasis and cholecystitis the site of pain is solely within the epigastrium.[129,130] In addition, when the chief complaint is right hypochondriac pain there is almost always some epigastric pain as well. There are reasons to believe that, regardless of its final radiations or other localizations, biliary duct pain (visceral pain unassociated with local peritonitis, adhesions, or other complications) is primarily epigastric. How often are unsuspected diseases of the gall bladder discovered during laparotomies? The fact that the anatomical area overlying the gall bladder itself is often not directly indicated by the symptoms is borne out by the considerable percentage of instances in which gallstones are clinically characterized as "silent." The records of these cases carry such statements as the following: "there were no preoperative symptoms referable to the gall bladder," "the complaints before operation were entirely gastric." In the absence of a typical biliary colic a diagnosis of "dyspepsia," "atonic gastritis," "chronic indigestion," is made with great frequency. Often it is only when a true "biliary colic" has occurred, with its right upper quadrant tenderness and rigidity and with the associated pain to the right shoulder, inspiratory dyspnea, and possibly jaundice, that attention is finally directed to the stone which for months or years had been causing varying degrees of epigastric discomfort or even, with the ingestion of fried, fat or greasy foods, of real epigastric pain. The so-called gastric symptoms of biliary tract disease are usually said to be due to a "reflex stomach" (viscero-visceral or vagus reflexes) and such gastric dysfunctions as hypermotility, hyperirritability, hypersecretion and pyloro-

spasm, may be demonstrated by fluoroscopic examination or the analysis of gastric contents; yet there seems clear evidence that in addition, or perhaps of even more importance

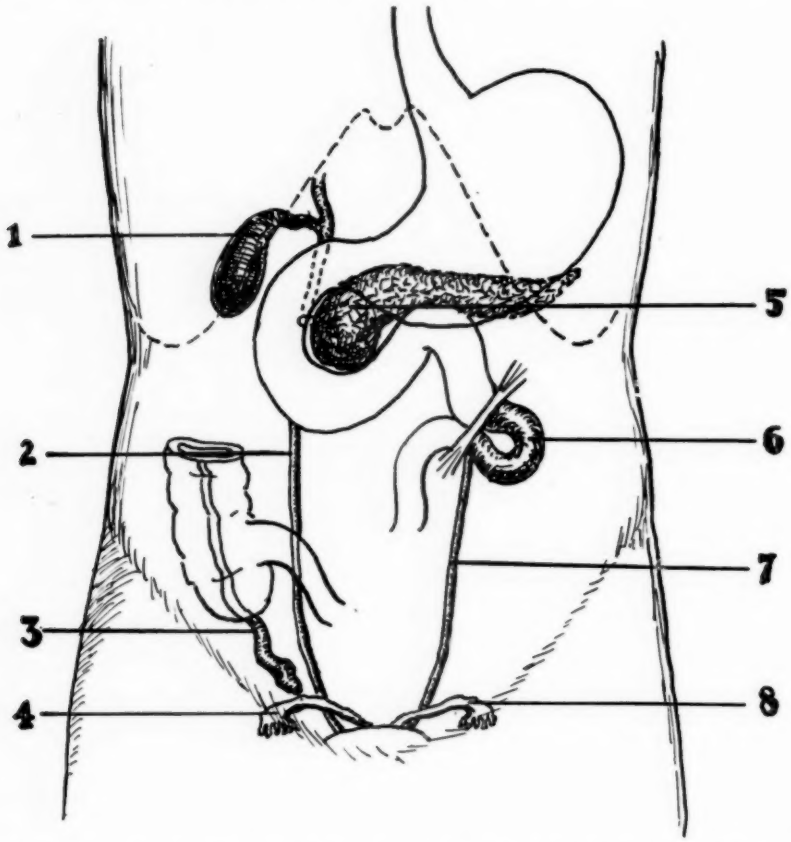

FIG. 96. Tubular structures often giving rise to colicky abdominal pains. (After Cope.)

1. Gall bladder and biliary ducts. 2. Right ureter. 3. Appendix. 4. Right fallopian tube. 5. Pancreatic ducts. 6. Loop of small bowel. 7. Left ureter. 8. Left fallopian tube.

in the production of epigastric distress, the gall bladder itself, without any reference to stomach dysfunctions, is capable of initiating epigastric pain.

The Nature of Colics

Colics occur almost exclusively in muscular tubes or in ducts of small caliber (Fig. 96), as biliary ducts, ureters, loops of small intestine, the characteristic intense intermittent pain being associated with strong peristaltic waves or smooth muscle spasms. Increases in intraluminary tension seem to be

an important factor in the causation of the typical colics. Since the stomach is a hollow muscular organ it might be anticipated that this organ would be the cause of pain of a cramp-like or colicky nature. Yet the typical gastric pain is not of this type and true colics from stomach affections are of rare occurrence. In his study of epigastric pain Mackenzie has gone so far as to state, "it will usually be found in the long run, that the so-called 'cramp in the stomach' in which there is pain of great severity referred to the epigastrium is due to gallstone colic. I have watched many patients for years who suffered from these severe attacks, and found that they *all* turned out to be cases of gallstone colic."[127] Colicky pain in the epigastrium, then, constitutes presumptive evidence of the presence of gallstones.

Gastric Pain Simulating Angina Pectoris

In those rare instances in which the stomach itself does undergo a sufficiently violent and massive contraction to cause pain of a colicky nature the resulting pain is not difficult to differentiate from the colics (right hypochondriac or epigastric) of biliary tract diseases. For when the stomach contracts as a whole there is implication of the cardiac as well as of the pyloric end of the organ and this causes pain which extends upward well over the sternum and to areas supplied by the fifth or even the fourth thoracic nerves. This pain not infrequently is associated with the regurgitation of acid gastric contents into the lower end of the esophagus and with the symptoms of pyrosis of heartburn. Because of the height to which it extends such pain has been mistaken for that of angina pectoris. Although the pain reference may be over the area of the heart the condition is but a pseudoangina for it is not related in any way to actual cardiac disease; pain, tenderness and hyperesthesia, such as extends into the arm with true angina, serve to distinguish the diseases and a complete study of the circulatory system combined with a good history should leave but little doubt as to the final diagnosis.

The characteristic pain of stomach lesions, particularly of ulcers, is dull, boring and deep-seated. It does not have either

The Pain of Peptic Ulcer

the intensity nor the spasmodic nature of a colic. In most instances it has a certain periodicity and its outstanding feature is a relation to the intake of food. While the time of onset of the pain after eating varies in individual cases, with the single case there tends to be an almost "clock-like" regularity in the onset of the discomfort after the taking of food.[131] A young individual who is hungry but afraid to eat because eating regularly sets up pain almost surely has a peptic ulcer. Ulcer distress is a quantitative dyspepsia rather than a qualitative dyspepsia as is seen with gall bladder diseases. The character of the food makes little difference; it is the fact that something is placed in the stomach which causes the distress; when the ulcer pain is severe even very small amounts of food uniformly initiate the symptom. The time of onset of pain bears a rough relationship to the distance of ulceration along the digestive tract. It has been stated that pain occurring immediately, or within a half-hour after eating, indicates ulceration near the esophagus, while that beginning from half-an-hour to an hour-and-a-half after the ingestion of the meal is probably indicative of a prepyloric lesion; and that when the postcibal pain is still later in onset the ulcer is likely to be duodenal.

Cycles or Rhythms with Peptic Ulcer Pains

This relationship to food has been termed "cyclic" in nature and Moynihan has spoken of a "rhythmicity" with peptic ulcer pain.[132] The rhythm with duodenal ulceration is triple and with gastric ulceration is quadruple. With duodenal ulceration the cycle is: food-relief-pain, food-relief-pain (a triple rhythm), for the discomfort begins so long after the ingestion of food as to be termed a "hunger-pain" and following the intake of food or alkalies there is a long period of relief. With gastric ulcer the rhythm is: food-relief-pain-relief; food-relief-pain-relief (a quadruple rhythm) for the pain wears off gradually before time has arrived for the ensuing meal and the period of relief after eating is so brief that the patient fears the effect from putting anything into the stomach.

These few clinical points relative to epigastric pains and to body-wall pains from gastrointestinal tract affections in

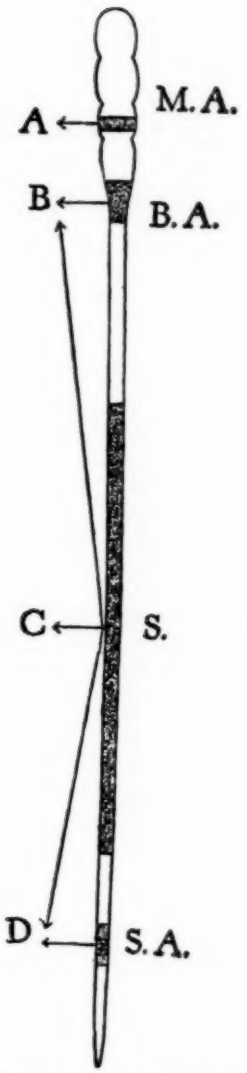

FIG. 97. Nerve supply of gastrointestinal tract. (From Langley.)

A. Sphincter of iris. Ciliary muscle.

B. Dialator of iris. Orbital muscle. Heart, Blood-vessels of mucous membrane of head. Walls of gut from mouth to descending colon. Outgrowths from this region of the gut (muscle of trachea and lungs; gastric glands, liver, pancreas).

C. The skin (arteries, muscles, glands). Blood-vessels of gut between mouth and rectum, of lungs and of abdominal viscera. Arteries of skeletal muscle. Muscle of spleen, ureter, and of internal generative organs. Walls of stomach, intestine, gall bladder and ducts, urinary bladder.

D. Arteries of rectum, anus and external generative organs. Walls of descending colon to end of gut. Walls of bladder and urethra. Muscle of external generative organs.

MA. Mid-brain autonomic.

BA. Bulbar autonomic.

S. Sympathetic. (I. Th.–II. or III. L. in man.)

SA. Sacral autonomic. (II.–IV. Sacral in man.)

Nerve Supply of the Digestive Tract

general have, perhaps, been rendered somewhat more readily understandable through the study of the development of the alimentary tube. A consideration of similar clinical rules regarding umbilical and hypogastric pains will be deferred in order that still other embryological arrangements, important in explaining everyday surgical matters, may first be investigated. A detailed review of the innervation of the gastrointestinal tract is reserved for a separate section (visceral neurology) where specific nerve connections and individual reflex pathways may be dealt with. Such additional study will make apparent that the reference of enteral pains to the tissues of the midline of the body wall takes place by way of the splanchnic nerves (sympathetic nervous system). The production of additional disturbances within the tissues of the body surface by way of the vagus nerves (autonomic fibers) will later be considered (reflex phenomena of scalp, temples, face, jaws, neck). The briefest summary of the nerve supply of the gastrointestinal tract might be stated as follows:

a. A supply from the sympathetic system (splanchnic nerves) to stomach, small intestine and the greatest part of the great intestine.

b. A supply from the bulbar autonomic system (vagus nerves) to the tract walls from the mouth to the descending colon.

c. A supply from the sacral autonomic (nervus erigens or pelvic vagus) to the descending colon and rectum.

d. A supply to the extreme cranial and caudal ends of the tract (derivatives of stomatodeum and proctodeum) from the cerebrospinal nervous system (Fig. 97).

EPONYM

JAMES BENIGNUS WINSLOW*

Eponym: Foramen of Winslow. Foramen epiploicum; a small orifice of communication between the greater peritoneal cavity and the omental bursa (lesser peritoneal cavity; Winslow's pouch).

Nativity: Odeuse, Denmark, April 2, 1669.

Death: April 3, 1760, aged ninety-one.

Education: First studied theology in native land.

1697: Pre-medical studies in Holland.

1698: Continued studies in Paris.

Student of famous French anatomist Duverney who encouraged his natural inclination toward anatomy.

1702: Matriculated in University of Paris.

1703: Delivered thesis dedicated to Bossuet (French writer and orator) who, being ill, had himself carried to the university to hear the presentation.

1705: Degree, Doctor of Medicine, University of Paris.

Connections: 1707: Presented to Royal Academy of Sciences by Duverney. Duverney had such confidence in his pupil that he often entrusted him with his lectures on Anatomy and Surgery at the Jardin du Roi. After the death of Duverney the chair at Royal Garden fell to Hunault, but later to Winslow. Winslow continued to fulfil this assignment as long as age permitted.

Writings: Eponymic not only with reference to the foramen epiploicum but also for Winslow's ligament (a fasciculus forming part of the posterior ligament of the knee joint ligamentum popliteum obliquum running from the tendon of the semimembranosus to the back part of the outer condyle of the femur) and for Winslow's stars (capillary whorls in the lamina choriocapillaris from which arise the venae vorticosae). Winslow did much to systematize the anatomical knowledge of his time, particularly in reference to myology.

Among his published books and papers were:

1711: Observations sur les fibres du coeur et sur les valvules, avec la manière de la préparer pour le démontuer.*Mem. de l'acad d. Sc.*

1715: Nouvelles observations anatomiques sur la situation et la conformation de plusieurs visceres. *Mem. de l'acad. d. Sc.*

1717: Description d'une valvule singuliere de la veine cave, et nouveau sentiment sur la fameuse Question du trouovale.

1729: Observations anatomiques sur la rotation, la pronation, la supination, et d' autres mouvements en rond.

1732: Exposition anatomique de la structure du corps humain. Paris, also English, German and Italian versions.

1742: Dissertation sur l'incertitude des signes de la mort. Paris.

Source of Eponym: Exposition anatomique de la structure du corps human, Paris, 1732, 2: 170, 352–365.

Discussion of Eponym: Winslow tested the communications of the foramen bearing his name by the ingenious method of inserting into this opening a quill or pipe, well fitted to the orifice, and then after blowing through the quill, observing the course and distribution of the air. He found that air distended the greato mentum, filling it like a bladder or balloon.

The surgeon seldom thinks of the great omentum as being "a bladder" and only sees the potential space within the omentum filled when, for example, loops of small bowel have passed through the foramen epiploicum (hernia through foramen of Winslow) or when blood or gastric contents occupy the cavity after injury to or perforation of

*See photostat regarding first name.

EXPOSITION
ANATOMIQUE
DE LA STRUCTURE
DU CORPS HUMAIN,

PAR M. WINSLOW, Docteur-Régent de la Faculté de Médecine de Paris, Interprète de la Langue Teutonique à la Bibliotheque du Roi, Ancien Professeur d'Anatomie & de Chirurgie au Jardin Royal, de l'Académie Royale des Sciences & Belles Lettres de Berlin.

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A

AN
ANATOMICAL EXPOSITION
OF THE
STRUCTURE
OF THE
HUMAN BODY.

BY JAMES BENIGNUS WINSLOW,

PROFESSOR OF PHYSICK, ANATOMY AND SURGERY, IN THE UNIVERSITY OF PARIS,

MEMBER OF THE ROYAL ACADEMY OF SCIENCES, AND OF THE ROYAL SOCIETY AT BERLIN, &c.

TRANSLATED FROM THE FRENCH ORIGINAL,

By G. DOUGLAS, M.D.

Illustrated with COPPER PLATES.

VOL. II.

The FIFTH EDITION, corrected.

THE NEW YORK LIBRARY ACADEMY OF MEDICINE

B

FIG. 98. A. Facsimile of title page of volume II of French edition of Winslow's work containing his description of the foramen epiploicum.
B. Title page of English translation of Winslow's work, by G. Douglas.

DEDICATION.

TO

Dr. JAMES DOUGLAS.

SIR,

WHAT I here take the Liberty to offer You, is already more Your own than mine. To You I owe all that I know, not only of the Science, but of the Style of Anatomy; and there is not one good Line in this Tranſlation, which is not the better for what You have taught Me, or procured Me Opportunities of learning. Theſe are very great Obligations to a Man reſolved to live by the Profeſſion of Phyſick; but they are very ſmall, when compared with others that You have laid Me under, without which I ſhould never have been in a Condition to live by any Profeſſion. Pardon this publick Acknowledgment of Favours which You deſigned to have kept always ſecret; and continue to believe Me with the utmoſt Sincerity, Gratitude and Reſpect,

SIR,

Your Moſt Dutiful,

Bow-Lane,
Sept. 5, 1732.

Moſt Obliged Brother,

G. DOUGLAS.

Fig. 99. Unusual dedication to English edition of Winslow's work, in which G. Douglas pay a beautiful tribute to his brother James Douglas.

Situation, Division, and Connexion of the Omentum.

352. The Omentum is a large, thin, and fine membranous Bag, furrounded on all Sides by numerous Portions of Fat, which accompany and even inveſt the ſame Number of Arteries and Veins adhering cloſely to each other.

353. The greateſt Part of it reſembles a Kind of flat Purſe, or a Sportſman's empty Pouch, and is ſpread more or leſs on all the ſmall Inteſtines from the Stomach to the lower Part of the Regio Umbilicalis. Sometimes it goes down to the lower Part of the Hypogaſtrium, and ſometimes it does not reach beyond the Regio Epigaſtrica. It is commonly plaited or folded in ſeveral Places, eſpecially between the Bands of Fat.

354. It is divided into a ſuperior and inferior, an anterior and poſterior, and a right and left Portion. The ſuperior Portion is in a Manner divided into two Borders, one of which is fixed along the great Curvature or convex Side of the Arch of the Colon, and the other along the great Curvature of the Stomach. The Commiſſure or Union of theſe two Borders on the right Side is fixed to the common Ligament or Adheſion of the Duodenum and Colon, and to the contiguous Parts of theſe two Inteſtines. That on the left Side is fixed to the longitudinal Sciſſure of the Spleen, to the Extremity of the Pancreas, and to the convex Side of the great Extremity of the Stomach. It is likewiſe fixed to the membranous Ligament, which ſuſtains the Ductus Cholidochus, and connects it to the Vena Portæ Ventralis.

355. Below theſe Adheſions the other Portions, that is, the anterior, poſterior, two lateral and inferior Portions, which laſt is the Bottom of the Sacculus Epiploicus, have commonly no fixed Connexions, but lie looſe between the Foreſide of the Cavity of the Abdomen and the Inteſtines. The anterior and poſterior Portions are generally called the Laminæ of the Omentum, but as that Term is ordinarily employed to expreſs the Duplicature of ſome compound Membrane, it would be more convenient to call them Folia, Alæ, or ſome ſuch Name.

Structure of the Omentum.

356. The Membrane of the Omentum is, through its whole Extent, made up of two extremely thin Laminæ joined by a Cellular Subſtance; the Quantity of which is very conſiderable along the Blood Veſſels, which it every where accompanies in broad Bands, proportioned to the Branches and Ramifications of theſe Veſſels. Theſe Cellular Bands are more or leſs filled with Fat according to the Corpulency of the Subject, and for that Reaſon I have called them Bands or Portions of Fat.

Little Omentum.

357. Beſides this large membranous Bag, which I name the great Omentum, there is another much ſmaller, which differs from the large one, not only in Size, but alſo in Figure, Situation and Connexion; and this I name the little Omentum. This ſmall Bag is fixed by its whole Circumference, partly to the ſmall Curvature of the Stomach, and partly to the concave Side of the Liver before the Sinus of the Vena Portæ, ſo as to ſurround and contain the prominent Portion of the Lobulus.

358. The little Omentum is thinner and more tranſparent than the other, and its Cavity diminiſhes gradually from the Circumference to the Bottom, which in ſome Subjects terminates in ſeveral ſmall Cavities or Foſſulæ more or leſs pointed. Its Structure is pretty much the ſame with that of the great Omentum, it being compoſed of two Laminæ, with a Mixture of the ſame Portions of Fat, which are conſiderably finer than the other.

359. We ſee from this Situation of the two Omenta, that in the Space left between the lower Side of the Stomach and upper Side of the Meſocolon, they have a very broad Communication with each other; ſo that if either of them contained in its Cavity any Fluid, that Fluid might readily get between the Stomach and Meſocolon, and ſo paſs into the other Bag; eſpecially when the Stomach is empty and conſequently its Situation eaſily changed.

360. Therefore by Means of this Interſtice between the Stomach and Meſocolon, the two Omenta form one Cavity, which opens into the Cavity of the Abdomen by one common Orifice, ſituated near the Commiſſure on the right Side of the great Omentum. This Orifice is ſemilunar or ſemicircular, and formed by the Union of two membranous Ligaments, whereof one connects the Beginning of the Duodenum and Neck of the Veſicula Fellis to the Liver; the other connects the contiguous Portion of the Colon to the ſame Viſcus, and extends to the Pancreas. From thence ariſes an incurvated Border, which ſurrounds the Root of the Lobulus, leaving an Opening wide enough to admit the End of the Finger.

361. To diſcover this Orifice of the Omentum, we need only raiſe a little the great Lobe of the Liver, and find out the Root of the Lobulus, and apply to it a large Pipe wrapt round with Cotton, Wool, or Tow, to hinder the Regreſs of the Air. Then if we blow gradually, the Air will inflate the Sides of the great Omentum, and give it the Appearance of a large Bladder irregularly divided into ſeveral Lobes or Tubercles by the Bands of Fat, which appear, in this State, like ſo many Frœna between the Lobes.

362. To be ſure of ſucceeding in this Experiment, the two Omenta muſt be in their natural State, and they muſt be handled very gently with the Fingers firſt dipt in Oil. It ſucceeds better in young, lean Subjects, than in old or fat Subjects.

363. When we touch theſe Membranes with dry Fingers, they ſtick to them ſo cloſely as hardly to be ſeparated without being torn, as we ſee by the reticular Holes which appear in thoſe Portions of the Membranes that have been thus handled. In that Caſe it is to no Purpoſe, to blow through the natural Orifice already mentioned; and it is owing to theſe ſmall Holes that the Membranes of the Omentum have been ſuppoſed to be naturally reticular.

364. The membranous Laminæ of the little Omentum are contiguous partly with the external Membrane of the Liver, partly with that of the Stomach, and a little with the Membrane that lines the neighbouring Portion of the Diaphragm. Thoſe of the great Omentum are continued partly with the ſame Coat of the Stomach, and partly with the external Covering of the Colon, and conſequently with the Meſocolon; and they likewiſe communicate with the Covering of the Spleen.

365. We may ſatisfy ourſelves concerning theſe Continuations, by making a ſmall Hole in one of the Laminæ of the Omentum near the Stomach, Colon, &c. and by blowing into that Hole, through a Pipe well fitted to it; for the Air will gradually inſinuate itſelf under the common Coats of theſe Viſcera: But if the Parts be dry, they muſt be moiſtened a little, before the Experiment is made.

FIG. 100. Facsimile of English translation of words of Winslow relative to the foramen epiploicum.

A. J. S., n. s. Vol. VIII, No. 6, p. 1360

the posterior wall of the stomach. At other times the great omentum resembles a caul or great apron overlying the small intestinal loops (flat). Winslow's observations in regard to distention of the great omentum by air may be verified, however, providing the experimenter moisten his fingers in oil as directed, when handling the parts.

Study of the original words of Winslow is rendered difficult for the modern student by reason of the fact that Winslow uses the term "lesser omentum" in a different sense from that in which it is now employed. The "lesser omentum" of this author corresponds to the omental bursa (lesser peritoneal cavity) of the present. When Winslow's writings are reviewed, substituting "omental bursa" for "lesser omentum," the descriptions become more readily intelligible.

Winslow seems to have been the first to employ the term omentum in its present sense. He describes the cavity of the great omentum as resembling a "sportsman's purse or bag"; and much might be said in favor of his method for describing the omental bursa, and for terming this space and its peritoneal coats a "lesser omentum." Thus a hand introduced through an opening in the gastrocolic or great omentum lies within the large "sportsman's pouch" whereas a hand introduced into an opening in the present lesser omentum (above the stomach) (through the gastrohepatic omentum) would lie in the smaller "sportsman's pouch." These two "pouches" would be found to communicate with each other, as described, and to open in common through the foramen epiploicum into the general or greater peritoneal cavity.

Points of Interest:

Although the photostat of the English Translation of Winslow's "Exposition Anatomique" shows the first name James, dictionaries and medical histories variably use the first names James, Jacob, and Jacobus.

In beginning his medical studies Winslow was granted financial aid or "a pension" by the government of Denmark, in order "that the country might not be without participation in the glory of advancing the progress of anatomical knowledge." Later he received financial aid from the Bishop of Meaux. The Bishop died in 1704 (one year prior to Winslow's graduation). Deprived of aid Winslow addressed himself to the faculty of the University of Paris "which was not deaf to his pleas but even paid all his expenses for the rest of his course."

Winslow's father and grandfather were Lutheran pastors and Winslow first studied for the ministry. He then turned to the study of medicine and finally forswore the religion of his fathers to turn Catholic. He was, therefore, disinherited by his family.

It has been written that Winslow's exposition on the structure of the human body was the "first treatise of descriptive anatomy divested of hypothetical physiological details and explanations foreign to the subject" and that his work represented a "close description derived from actual objects rather than from reference to the writings of previous anatomists."

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QUESTIONNAIRE

1. What is the mechanical effect of the loss of abdominal fat?
2. Name some of the functions of intra-abdominal fat.
3. Why does it often prove so difficult to reduce a large umbilical or ventral hernia?
4. What special preoperative care is advisable in treating fat patients who have large ventral hernias?

5. What is meant by static pressure?
6. Explain some of the interrelationships between intravisceral, intraperitoneal and static pressures.
7. Discuss the effects of gravity in regard to the Fowler position, the Trendelenburg position, the knee-chest position.
8. What is meant by the cushion effect of air-containing organs?
9. What is the effect of an increase in the pressure exerted upon an organ containing fluid; one containing gas?
10. Explain the effects of gravity upon hernial masses.
11. Why do organs herniate both downward through the inguinal rings and upward through the diaphragm?
12. What is meant by garrulity of the vulva? Give the mechanism by which this phenomenon is produced.
13. What is the value of the knee-chest position?
14. State the instructions to be given to a woman who is to take the knee-chest exercise.
15. What is the habitus enteropticus?
16. What is visceroptosis?
17. What is the intra-abdominal condition found to be present with a congenital visceroptosis?
18. Why do patients with visceroptosis have sharp intercostal angles?
19. What are the chief symptoms of visceroptosis? Their cause?
20. State some of the methods for replacing and retaining in position prolapsed abdominal viscera.
21. What are some of the causes of acquired visceroptosis?
22. What are the chief differences as to symptoms and treatment between congenital and acquired visceroptosis?
23. What is the usual effect of pregnancy upon the symptoms of visceroptosis?
24. Give the technic of one or more methods for supporting the abdomen by means of adhesive dressings in the treatment of visceroptosis.
25. What requirements must be fulfilled by an abdominal belt as used in the treatment of visceroptosis?
26. What exercises are of particular value in the treatment of visceroptosis? How do these act to prove the condition of the patient?
27. Give a list of from 5 to 10 factors which have an important bearing upon pressure conditions within the abdomen.
28. Is the peritoneal cavity an actual or a potential space?
29. What effect has opening the peritoneal cavity upon the tensions between the various abdominal viscera and between the viscera and the abdominal walls?
30. When is it correct to use the term intra-abdominal pressure? What are the objections to the term?
31. Of what importance is it in instituting treatment to correctly interpret among the many forces active, the actual cause or causes of any abnormal pressure conditions within the abdomen?
32. What is the length of the alimentary canal? What factors seem to necessitate this extreme length?
33. What is the derivation of the following terms; intestine, alimentation, digestion?
34. From what primary germ layers are the following tissues derived; inner lining of enteral canal, skin, peritoneum, nerves of intestinal tract, muscles of intestinal walls?
35. What is meant by the following terms; yolk sac, yolk vesicle, vitelline duct, omphaloenteric canal, omphalomesenteric vessels, Meckel's diverticulum, archenteron?

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36. What is the primary loop of bowel; name the parts of this loop. What is the foregut, hindgut, midgut?
37. Why does a loop of gut protrude into the umbilical cord in early embryonic life?
38. Explain with sketches the embryological development of the gastrointestinal tract.
39. What is the normal incidence of a persistent omphaloenteric duct?
40. What are the various shapes in which Meckel's diverticula may occur?
41. What is the usual length of a Meckel's diverticulum? What is the average distance from the ileocecal valve for the attachment of the diverticulum to bowel? How much variation is there as to the site of this attachment?
42. What vessels may accompany a Meckel's diverticulum? What are the origin and function of such vessels?
43. In what ways do acquired diverticula of bowel differ from congenital diverticula?
44. Contrast acquired and congenital diverticula on the following points; number, shape, size, position, structure, age incidence, potentialities.
45. What is a false diverticulum?
46. Name several ways in which a Meckel's diverticulum or persistent omphalomesenteric vessels may cause intestinal obstruction.
47. What is a diverticular knot? Sketch some of the more common types of congenital diverticula and the more common intra-abdominal complications they may cause.
48. Why are pains from gastrointestinal affections most often referred to the midline of the anterior abdominal wall?
49. To what areas are pains referred from affections of the following structures; esophagus, duodenum, biliary ducts, ileum, jejunum, cecum, appendix, colon, rectum? Why do these cause pains limited to the areas described?
50. Where is the primary pain of the following conditions most likely to be referred; volvulus of sigmoid colon, herniation of small bowel into fossa of Treitz, gastromesenteric ileus, biliary colic, duodenal ulcer, gastric ulcer, acute esophageal dilatation, diaphragmatic pleurisy, appendiceal colic, Deitl's crisis, embolism of superior mesenteric artery?
51. What evidence is there that pains due to visceral affections are referred to skeletal tissues and that the pain is not actually referred to the involved viscus itself?
52. What are the chief zones found by Mackenzie as the sites for referred pains with gastrointestinal affections?
53. What accounts for the inequality between the length of the body wall zones to which pain is referred from various units of the alimentary canal and the length of the bowel divisions themselves?
54. Why is large bowel pain felt at a lower point than small bowel pain when the large bowel reaches to the hypochondriac regions while the small bowel lies in part within the pelvis?
55. At what period in development are nerve pathways in the gastrointestinal tract determined? Do these early central nerve connections change in later life? If so, how?
56. What percentage of gastric symptoms are due to actual gastric lesions?
57. What are the chief extragastric causes of gastric symptoms?
58. What is meant by terming the stomach "the abdominal spokesman?"
59. How do the following conditions cause alterations in gastric motility and sectretory activity; brain tumor, basilar meningitis, cholelithiasis, tabes mesenterica, Pott's disease?
60. Along what broad lines are gastric lesions to be differentiated from extragastric conditions causing gastric symptoms?
61. What is meant by "silent gallstones?"

62. What percentage of the distress with cholelithiasis and cholecystitis is epigastric in location?
63. Describe a typical biliary colic. What are the chief associated symptoms?
64. What is meant by a "qualitative dyspepsia;" a "quantitative dyspepsia?"
65. What are the characteristics of a colicky pain? Causes?
66. What is the usual cause of a severe epigastric colic?
67. What is the site of the colicky pain due to a massive or generalized contraction of the stomach?
68. How may gastric affections simulate angina pectoris?
69. What is the typical pain with peptic ulcerations?
70. Explain the relationship between ulcer pain and the ingestion of food.
71. What is meant by a "rhythm" or "cycle" in ulcer pain?
72. What is meant by the "triple rhythm of duodenal ulcer pain" and the "quadruple rhythm of gastric ulcer pain?"
73. By what nerve pathways are pains of gastrointestinal tract origin referred to the forehead, scalp, jaw and neck?
74. Briefly describe or sketch the innervation of the gastrointestinal tract.

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